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GROWTH OF THE CHILD AND THE CALCIFICATION PATTERN OF THE TEETH

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INTRODUCTION

IT IS well known that the development of the hard tissues of the body (enamel, dentin, and bone) are affected by disturbances in the metabolism of the individual. Such disturbances are readily recognized by differences in the quality of the calcification of the hard tissues. The purpose of this paper is, first, to analyze the differences in the quality of the enamel and dentin formed and calcified during the various developmental periods of the growing individual; and second, to attempt to correlate these differences with the physiologic characteristics of those periods. The knowledge thus obtained by the use of the tooth as a biologic indicator should be of value to the pedodontist, the orthodontist, and the pediatrician.

This paper is also designed to serve as a basis of "normality" or "average" quality of tooth structure against which current studies of the effects of diet and endocrines upon the quality of tooth structure can be analyzed.

REVIEW OF LITERATURE

A review of the literature shows that comparatively few studies have been made to determine accurately the quality of tooth structure during different age periods. The work of Mellanby,[1] Swanson,[2] Karnosh,[3] Schour,[4] and Massler, Schour, and Poncher[5] represent the major endeavors in this direction. Table I summarizes their observations.

From the Child Research Clinic and Department of Histology, University of Illinois College of Dentistry.

TABLE I. COMPARATIVE DATA FOR PERIODS OF GOOD AND POOR CALCIFICATION OF DECIDUOUS AND PERMANENT TEETH

TEETH*	LEVELS EXAMINED	QUALITY OF CALCIFICATION OF ENAMEL		AGE PERIOD ACCORDING TO MELLANBY AND SWANSON	CORRECTED AGE PERIOD
		MELLANBY[1] AND SWANSON[2]	MASSLER, SCHOUR AND PONCHER[5]		
I II III / a b c	Incisal 2/3	Good	Excellent	Inception of calcification to about 6 months in utero	4 months in utero to birth
IV V / d e	Cuspal 1/3				
I II III / a b c	Cervical 1/5	Poor	Very poor	6 months in utero to 4 to 6 months after birth	Birth to about 10 months of age
IV V / d e	Middle & cervical 2/3				
6 / 6	Occlusal 1/3				
6 / 6	Middle 2/3	Good	Good	4 to 6 months to about 2 years of age	10 months to about 2½ years of age
1 3 / 1 2 3	Narrow band between incisal and middle 1/3†				
2 4 5 7 / 4 5 7	Incisal 1/3 Cuspal area				
6 / 6	Cervical 1/5	Poor	Poor	3 to 5 years of age	About 2½ to 5 years of age
1 2 3 / 1 2 3	Cervical 1/2				
4 5 7 / 4 5 7	Middle 2/3				
3 7 / 3 7	Cervical 1/5	Good	Fair	6 to 10 years of age	6 to 10 years of age
8 / 8	Occlusal 1/2				
8 / 8	Cervical 1/3	Poor	Poor	About 10 to 13 years of age	About 10 to 13 years of age

*In the dental formula permanent teeth are indicated by Arabic numerals, maxillary deciduous teeth by Roman numerals, and mandibular deciduous teeth by letters.

†Both Mellanby and Swanson included in this period the development of the cuspal enamel of the anterior teeth, and they therefore begin this period at 4 to 6 months of age. We begin this period at 10 months of age, on the basis of observations on the upper lateral incisor, which begins to form at about that time.

The Incremental Growth Pattern of the Enamel and Dentin and Its Chronology.—It is apparent that a thorough acquaintance with the incremental growth pattern of the enamel and dentin and its exact chronology is a necessary prerequisite to this study. Without such equipment, the analysis must necessarily be only an approximation. The incremental growth pattern and its chronology was therefore investigated and reported in a previous issue of the *Journal of the American Dental Association.*[6]

MATERIAL

The laboratory of histology of the University of Illinois College of Dentistry has for some time received teeth, deciduous and permanent, from various parts of the country. The bulk of the material was obtained from several sources in Chicago: dental clinics, and private practitioners and parents, who cooperated by saving all the shed or extracted teeth of their patients or children. Most of the teeth were accompanied by medical histories of the patients.

This study is based on about 1,000 human deciduous and permanent teeth from normal, healthy children, which were studied in ground and decalcified sections. The teeth were distributed evenly among the deciduous and permanent dentitions, and incisors, cuspids, and molars were fairly evenly represented. The decalcified sections were usually stained with hematoxylin and eosin.

The data on the incidence of hypoplastic defects herein recorded were those obtained from observing patients regularly admitted to the dental clinics of the University of Illinois and to the hospital dental clinic in the Research and Educational Hospital.

METHOD OF EVALUATING THE QUALITY OF ENAMEL AND DENTIN

All sections were examined under transmitted light. The quality of the enamel and dentin was judged by the homogeneity of the structure and the density of the calcification.

Enamel.—The density of the calcification of the enamel was judged by its degree of translucency and whiteness. Areas that appeared dark or black were regarded as poorly calcified. The number of incremental lines was used as another index of calcification, the greater their number, the lesser the degree of calcification. Swanson[2a] recognized that the striae of Retzius "range in a graded series from scarcely perceptible striations to relatively grave lesions, and reflect, by their magnitude and number, the changes that took place in the salt balance of the body as enamelization proceeded."

Hypoplasia of the Enamel.—If a systemic disturbance is sufficiently severe, not only the calcification of the matrix but the cells active at the time in the deposition of the matrix will be affected. The epithelial enamel-forming cells are much more sensitive to systemic interferences than are the mesenchymal dentin-forming cells. Whereas a severe systemic disturbance may be reflected in the presence of interglobular dentin in the dentin, the enamel-forming cells are often injured by the same effect and in such a manner that hypoplastic enamel results. Hypoplastic effects can thus be taken to indicate also severe disturbances in calcification. A consideration of the *incidence* of enamel hypoplasia is therefore included in this study because such formations reflect periods of severe systemic imbalance or of high cellular susceptibility to systemic interferences.

Dentin.—Variations in the degree of calcification of the dentin are more extensive and therefore more easily and objectively graded (Fig. 1). While the enamel is more sensitive than is the dentin to minor fluctuations in cal-

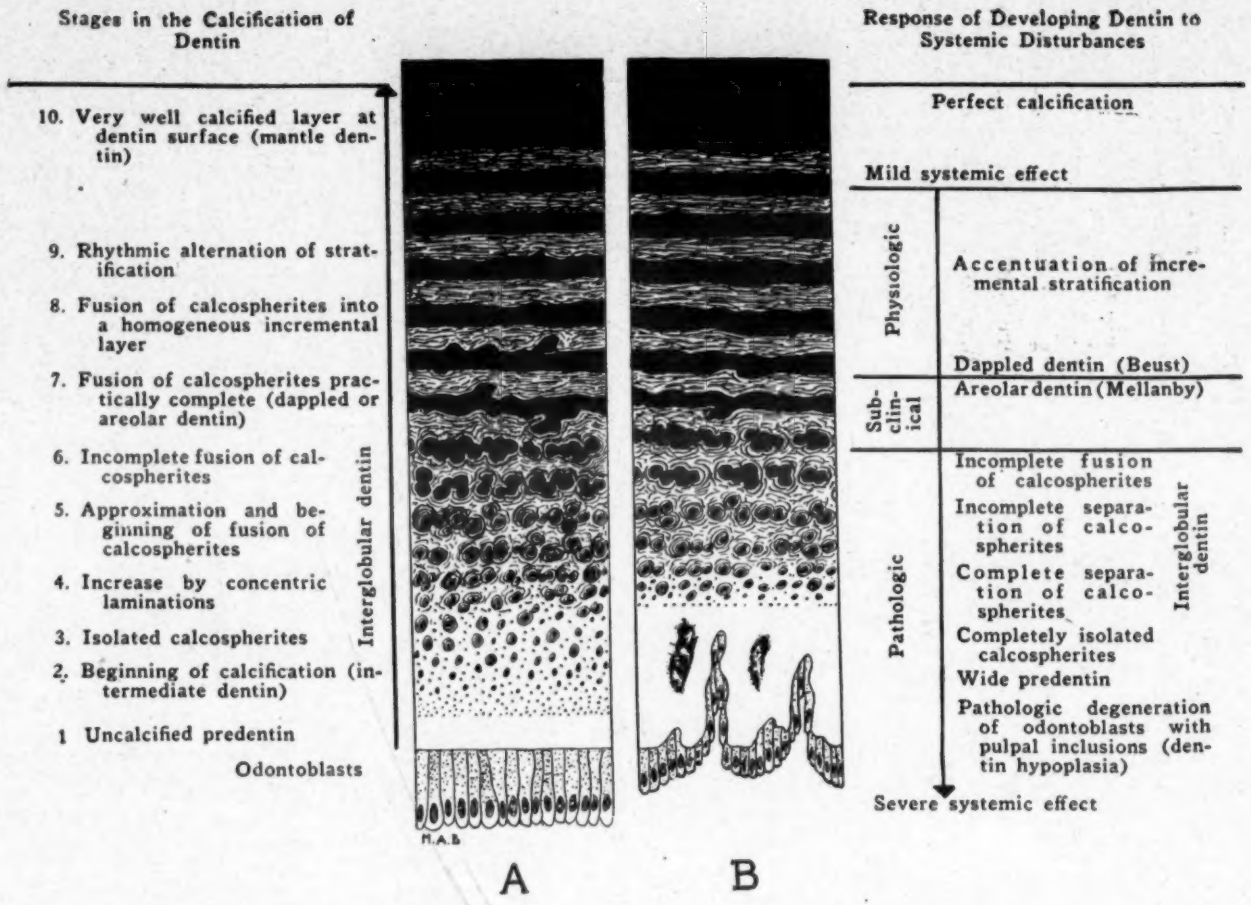

Fig. 1.—*A*, Stages in the calcification of dentin. *B*, Effects of systemic disturbances upon the calcification of the developing dentin. (From Am. J. Dis. Child. 62: 33-67, 1941.)

TABLE II. RESPONSE OF DEVELOPING DENTIN AND ENAMEL TO SYSTEMIC DISTURBANCES

SYSTEMIC EFFECT	REACTION IN DENTIN	REACTION IN ENAMEL
Mild		Accentuation of incremental lines (bands of Retzius)
	Accentuation of incremental stratification (Owen's lines of contour)	
		Increased number of striae and pigmentation
	Dappled dentin (Beust)	
	Areolar dentin (Mellanby)	
		Hypocalcification (mottled, chalky enamel)
	Interglobular dentin	
	Isolated calcospherites Wide predentin	(Ameloblasts show pathologic degeneration)
		Lines of arrested or deviated growth
	(Odontoblasts show pathologic degeneration)	Hypoplastic defects
	Lines of arrested or deviated growth	
	Hypoplastic defects (pu.pal inclusions)	
Severe		

cium metabolism, its range of reaction is more limited. The dentin can respond to such fluctuations in a greater variety of ways and therefore depicts the different degrees of fluctuation more accurately. (Table II.)

The severity of a given systemic interference is reflected in the quality of the calcification—the more severe the disturbance the more incomplete the process of calcification (Fig. 1). The basis for evaluation of the degree of calcification in the dentin may be summarized as follows:

1. A smooth, homogeneous calcification showing no incremental lines is considered good.

2. The appearance of the normal incremental stratification is considered to be within the normal physiologic variations of the process of calcification.

3. The imperfect fusion of the calcospherites, which gives a granular appearance to the dentin (dappled dentin of Beust and areolar dentin of Mellanby), is considered to indicate a subclinical deficiency in calcification. The presence of interglobular dentin, or the incomplete fusion of the calcospherites, is considered to be an indication of a marked disturbance in calcification. This interglobularity is subdivided into four degrees, which indicate the severity of the disturbance in calcification (Fig. 1): (a) incomplete fusion of the calcospherites, which gives to the dentin a distinctly mottled appearance; (b) complete separation of the calcospherites; (c) complete isolation of the individual basophilic calcospherites in a field of acidophilic, eosin-staining, uncal-

cified dentin matrix, and (d) usually associated with this, a wide predentin border of uncalcified dentin.

4. Prominent and acute accentuation of any one or several incremental lines is considered to indicate an acute disturbance in calcification and generally an arrest in matrix formation.

5. Death of the dentin-forming cells in localized areas results in the formation of pulpal inclusions, or hypoplastic defects. These are rarely seen and occur only in cases of the most severe systemic disturbances, such as prolonged parathyroprivic tetany.

FINDINGS AND DISCUSSION OF FINDINGS

Prenatal Calcification

Calcification begins in the deciduous teeth at the age of 4 to 6 months in utero. The incisal four-fifths and the occlusal one-third of the crowns of all the deciduous teeth were therefore examined (Figs. 2 and 3). In some instances the first permanent molars showed a small amount of prenatal calcification immediately over the mesiobuccal growth center or dentin cusp.

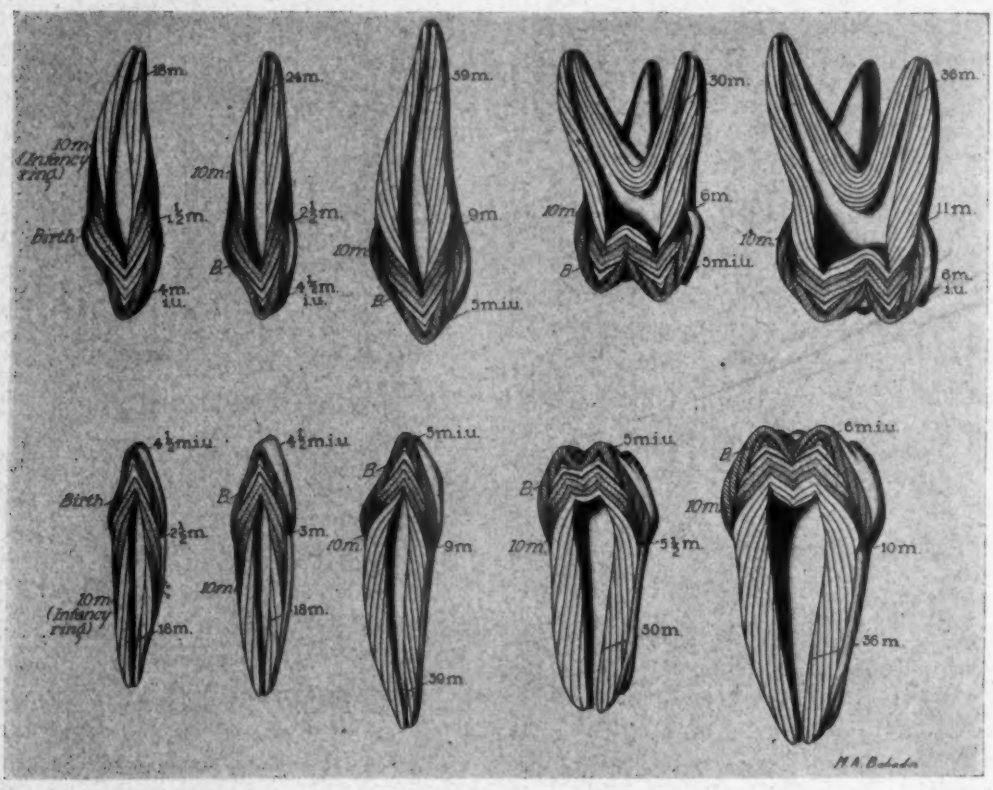

Fig. 2.—Diagrammatic representation of the calcification pattern superposed on the incremental pattern of the deciduous teeth in labiolingual sections. On the right of each tooth is indicated the approximate age at the beginning of tooth formation, at the completion of the crown, and at the completion of the root. Each increment represents approximately three months of tooth development. On the left of each tooth is indicated the level of the neonatal ring (birth) and the infancy ring (10 months). The infancy period (birth to 10 months) is stippled. In this figure *m* signifies months, and *m.i.u.*, months in utero. (From Am. J. Dis. Child. 62: 33-67, 1941.)

Findings.—Enamel: The enamel formed before birth is characteristically white and translucent. The calcification appears to be homogeneous and dense, relatively few incremental bands being present.

Dentin: The dentin is also homogeneously and densely calcified. Few incremental lines were observed. Less than 10 per cent of the teeth showed a slight increase in the prominence of the normal incremental stratification. In only two cases was any abnormal disturbance in prenatal calcification observed.

Hypoplastic defects: Hypoplastic defects in tissue calcified before birth were not found in our histologic material, but four children with such defects were observed among the children of the Orthopedic Hospital over a period of five years.

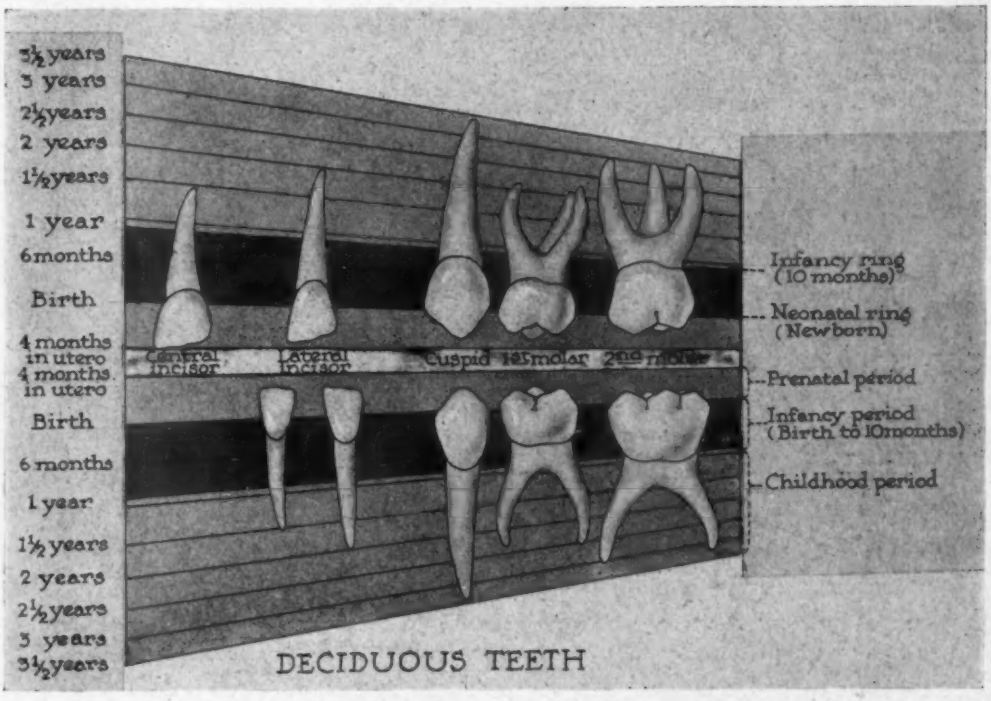

Fig. 3.—Chronology of development of the deciduous teeth in gross labial view. The horizontal lines are inclined to conform with the anteroposterior growth gradient of the teeth. The infancy period is shaded.

Review of Literature.—Mellanby,[1] Swanson,[2] and Karnosh[3] have agreed that the calcification of the enamel formed before birth is better than that of the enamel formed after birth. On the basis of an analysis in which the Nicol prism method was used, Rushton[7] concluded that the calcification of the enamel formed before birth is not homogeneous and is not of a higher quality than that of enamel formed after birth by the same ameloblasts.

Hypoplastic defects in the enamel formed before birth are rare. Wolfe[8] and Maxwell[9] described several cases of fetal rickets in which there were marked disturbances in such tissues. In the four cases of hypoplastic defects in enamel formed before birth which we have observed clinically, the condition was caused by marked subnutrition and toxemia of the mother during pregnancy.

Discussion.—The almost perfect calcification of the tissues calcified before birth is not surprising when one remembers that the embryo develops in an extremely well-protected and favored environment. The embryo or fetus is a parasite, deriving all of its nourishment from the mother and drawing on her calcium reserves in the bones when necessary.

The statement is frequently made that the maternal diet, particularly the amount of calcium (in the form of milk) ingested, is important in determining the quality of the tooth structure formed in utero. In our material, with but very rare exceptions, we could observe no difference in the quality of the enamel and dentin in the teeth from children of high or low economic groups. In fact, when the maternal diet was known to be deficient, examination of the teeth of the child both clinically and microscopically revealed no observable defects in the quality of the enamel and dentin. This may be correlated with the fact that in all but the most extreme deficiency states the maternal *reserves*, particularly of calcium, supply the tooth-forming material to the fetus.

In about 20 per cent of our material an accentuated ring was observed in the enamel and dentin at the chronologic level of about 7 months in utero. Sontag[10] also observed such a ring in bone. We have been unable to explain its existence.

Neonatal Ring

Findings.—The deciduous teeth and the mesiobuccal cusp of the first permanent molars regularly show an accentuated incremental ring at the level of the enamel and the dentin which is forming and calcifying at about the time of birth (Figs. 2 and 4). The presence of the ring can be demonstrated in virtually all deciduous teeth if sections are carefully prepared in a sagittal plane. It is much more readily apparent in the enamel than in the dentin. It appears dark or hypocalcified in the enamel and light in the dentin. This ring probably represents a line of arrested growth rather than a line of disturbed calcification and may be compared with the arrest lines often seen at the growing epiphyseal ends of the long bones.

Hypoplastic defects: Hypoplastic defects in the neonatal ring were found in only a few cases. These were analyzed and reported by Schour and Kronfeld.[14]

We estimate that about 78 per cent of all hypoplastic defects have their inception at birth and continue through the infancy period. About 3 per cent of these are acute and are confined to the neonatal period. They are seldom seen clinically, however, since the lesions are tiny and in the deciduous anterior teeth are often hidden by the gingivae (Fig. 6).

Review of Literature.—Rushton[11] and Sachs[12] reported the presence of a prominent contour line in the enamel formed at about the time of birth. Rushton[11] called it a "birth" line. Its physiologic significance and usefulness in dental investigative work, however, were not fully recognized until it was independently rediscovered by one of us (I. S) in 1936 and named the neonatal

line or ring.[4] The neonatal ring has since been used as a biologic landmark in investigations on the rate of formation of human enamel and dentin (Schour and Massler[13]) and on the effects of neonatal trauma on the teeth (Schour and Kronfeld,[14] Kronfeld and Schour[15]).

Discussion.—The neonatal line is the result of the brief arrest in growth subsequent to birth and reflects the physiologic readjustments incident to birth. It is not surprising that the profound metabolic changes which the newborn infant must undergo should leave their mark on the developing teeth. The loss in weight during the neonatal period and the temporary subnutrition are well-known manifestations of the same change. Under ordinary conditions the newborn infant does not regain its birth weight until the end of the first ten days of extrauterine life.

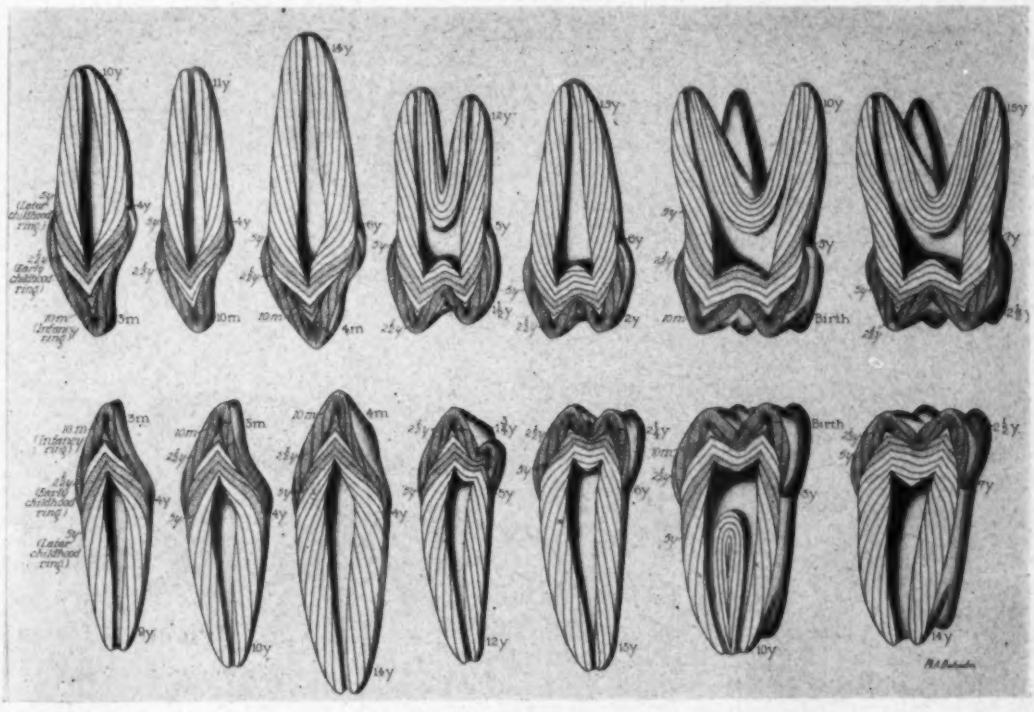

Fig. 4.—Diagrammatic representation of the calcification pattern superposed on the incremental pattern of the permanent teeth in labiolingual sections. Each increment represents approximately one year of tooth development. On the right of each tooth is indicated the approximate age at the beginning of tooth formation, at the completion of the crown, and at the completion of the root. On the left of each tooth is indicated the level of the neonatal ring (birth), the infancy ring (10 months), the early childhood ring (2½ years), and the later childhood ring (5 years). The infancy and later childhood periods are stippled in the dentin. In this figure, *m* signifies months, and *y*, years. (From Am. J. Dis. Child. 62: 33-67, 1941.)

Changes in bone during the neonatal period have been described (Harris,[16] Sontag[10]). Sontag[10] described a neonatal line in the round bones of the feet of infants. He found in some cases a "double" neonatal line in the long bones similar to that often observed in the teeth. The significance of the double effect is not known.

Hypoplastic defects: Pathologic accentuations of the neonatal ring depend on the severity of the adjustments during the neonatal period. They have been observed in cases of birth injury.[14] These accentuations may take the form of rings of greater width or deviations in the path of cellular activity, or they may be hypoplastic defects. That hypoplastic defects would occur at this time might be expected in view of the large number of pathologic conditions which constantly arise as a result of the trauma of birth. Birth injuries of premature infants are particularly likely to produce hypoplastic defects of the neonatal ring (Stein[17]).

The neonatal ring is present at the characteristic level even in the teeth of children born by cesarean section, which indicates that normally the neonatal ring results from the change in environment rather than from the type of birth. In premature infants the neonatal ring is found at an earlier chronologic level. A greenish coloration of the neonatal ring occurs in the deciduous teeth of children who have had a history of neonatal jaundice.[18]

Calcification During Infancy

Infancy is designated as the period extending from the end of the neonatal period to about the tenth month of age. We use the tenth month of age rather than the end of the first year as the termination of this period for reasons which will appear later. The levels of the enamel and the dentin of the deciduous and the permanent teeth forming during this period are shown in Figs. 2 to 5. Note that the upper lateral incisor, the premolars and the second and third molars have not yet begun their formation. The lag in the beginning of formation of the upper lateral incisor was pointed out by Zsigmondy[19] in 1894. It begins its formation at about the tenth month, at the end of the infancy period, and is therefore a valuable diagnostic aid in the estimation of the period at which hypoplastic defects were formed.

Findings.—The calcification of the dental tissues during the postnatal period is definitely less homogeneous than during the prenatal period. Accentuation of the incremental stratification in both the enamel and the dentin is the rule rather than the exception. This is seen most strikingly in the deciduous teeth, in which the calcification during the two periods (prenatal and postnatal) can be compared in the same section and is demarcated by the neonatal ring.

The decrease in the quality of the postnatal calcification becomes more marked as the age increases. Thus, the decrease in the quality of the calcification is only slight during the first three months but becomes increasingly more prominent from the third to the tenth month. At the tenth month an abrupt recovery in the quality of the calcification frequently occurs. This is true not only of the teeth of normal children; it is accentuated in those of children suffering from disturbances during infancy which cause marked interglobular dentin and hypoplastic defects in the enamel. A complete and sudden recovery from these defects usually occurs at the tenth month, although the illness and its clinical manifestations may persist.

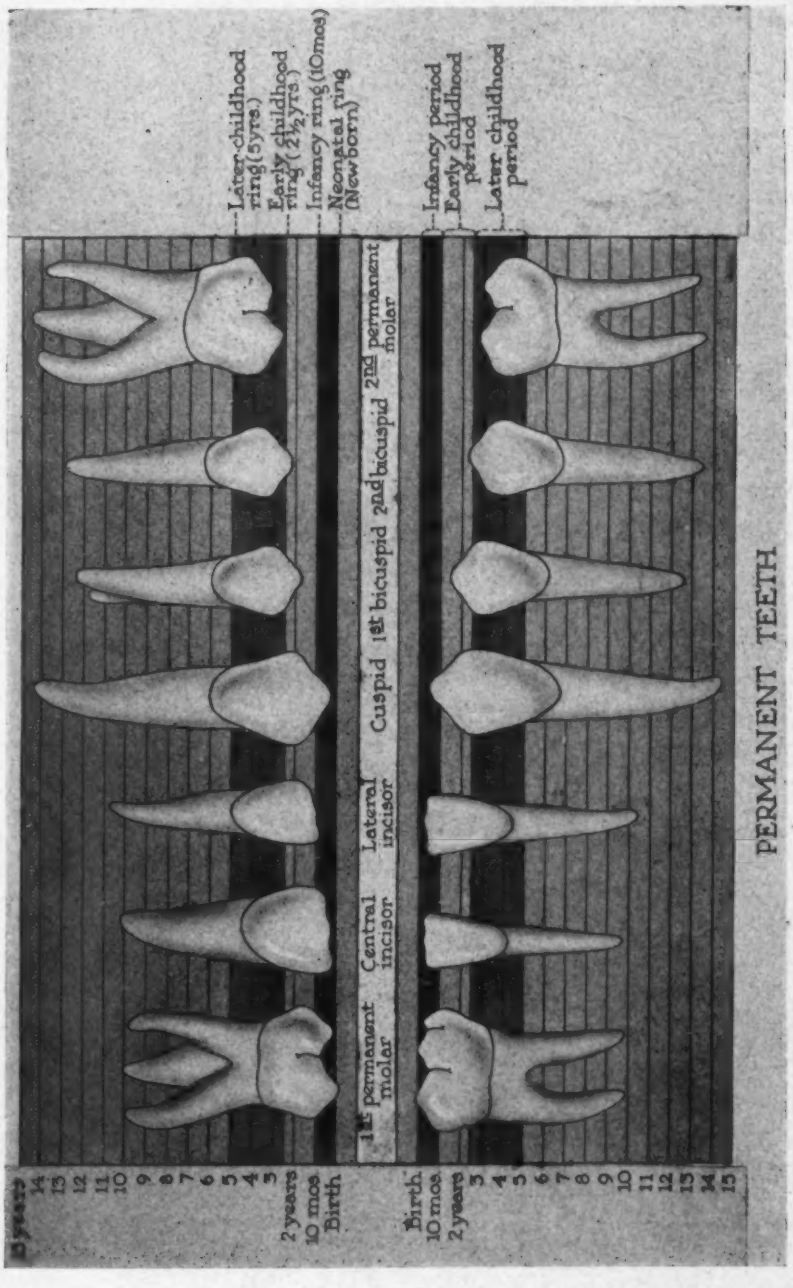

Fig. 5.—Chronology of development of the permanent teeth. The levels of the developmental rings and periods are indicated on the right. The infancy and later childhood periods are shaded. The first permanent molar is placed first since, chronologically, it is the first permanent tooth to develop.

During our analysis another fact became evident. In a group of teeth obtained from practitioners whose patients were limited to the well-to-do classes, the quality of the calcification during infancy, although poorer than that occurring in the prenatal period, was definitely better than the calcification in a second group of teeth obtained from a free extraction clinic, which served the poorer sections of Chicago. In the latter group the postnatal calcification was almost invariably poor, interglobular dentin and accentuated incremental lines in the enamel being the rule. This is in direct contrast to the fact that the *prenatal* calcification in both groups was practically identical. Mellanby[1b] has observed such differences in the postnatal calcification of teeth obtained from private sources and from dental clinics.

Hypoplasia of the enamel: The incidence of hypoplastic defects in the enamel and marked interglobular dentin was found to be highest during the infancy period. Sixty per cent of the total hypoplastic defects that occurred in our material were found to begin at birth and to extend through the infancy period. These are therefore chronic hypoplastic defects (Sarnat and Schour[20]). At the age level of the tenth month, the hypoplastic defects always showed a marked accentuation followed by complete recovery. *Usually these defects ended abruptly at 10 months of age, indicating not a sudden recovery from the systemic disturbance but rather a difference in the metabolic and cellular response of the tissues at that age, since our records generally showed that no change had occurred in the clinical condition of the patient.*

The finding of hypoplasia of the infancy period is facilitated by the fact that, in most instances, the upper lateral incisors are not involved since their formation and calcification begin at about 10 months of age (Fig. 6). Thus hypoplastic defects ending at the tenth month leave the upper lateral incisors untouched. Occasionally the upper lateral incisors begin to form at about the ninth month. In such cases, the tips (*mamelons*) of the upper lateral incisors are also affected by the hypoplasia of the infancy period.

Review of Literature.—Further evidence of a "normal" deficiency in calcification during infancy has been found in the growing bones of normal children. Robinson[21] examined the growing epiphyses of 240 healthy infants radiographically and found some evidence of rickets in 60.8 per cent of the children. Of these, 33 per cent showed positive and 27.8 per cent showed "questionable" evidence of rickets. The incidence of rickets dropped markedly after nine months of age.

The frequency of rickets and tetany during infancy is well known and results from the increased demand for calcium and phosphorus brought about by the rapid rate of growth. The rate of growth influences the localization of the manifestations of disease. Thus, actively growing areas, such as the epiphyseal ends of the long bones, are more apt to reflect diseases affecting growth (rickets, scurvy, and syphilitic osteochondritis) than are the areas which grow slowly or not at all. Eliot and Park[22] stated the problem succinctly:

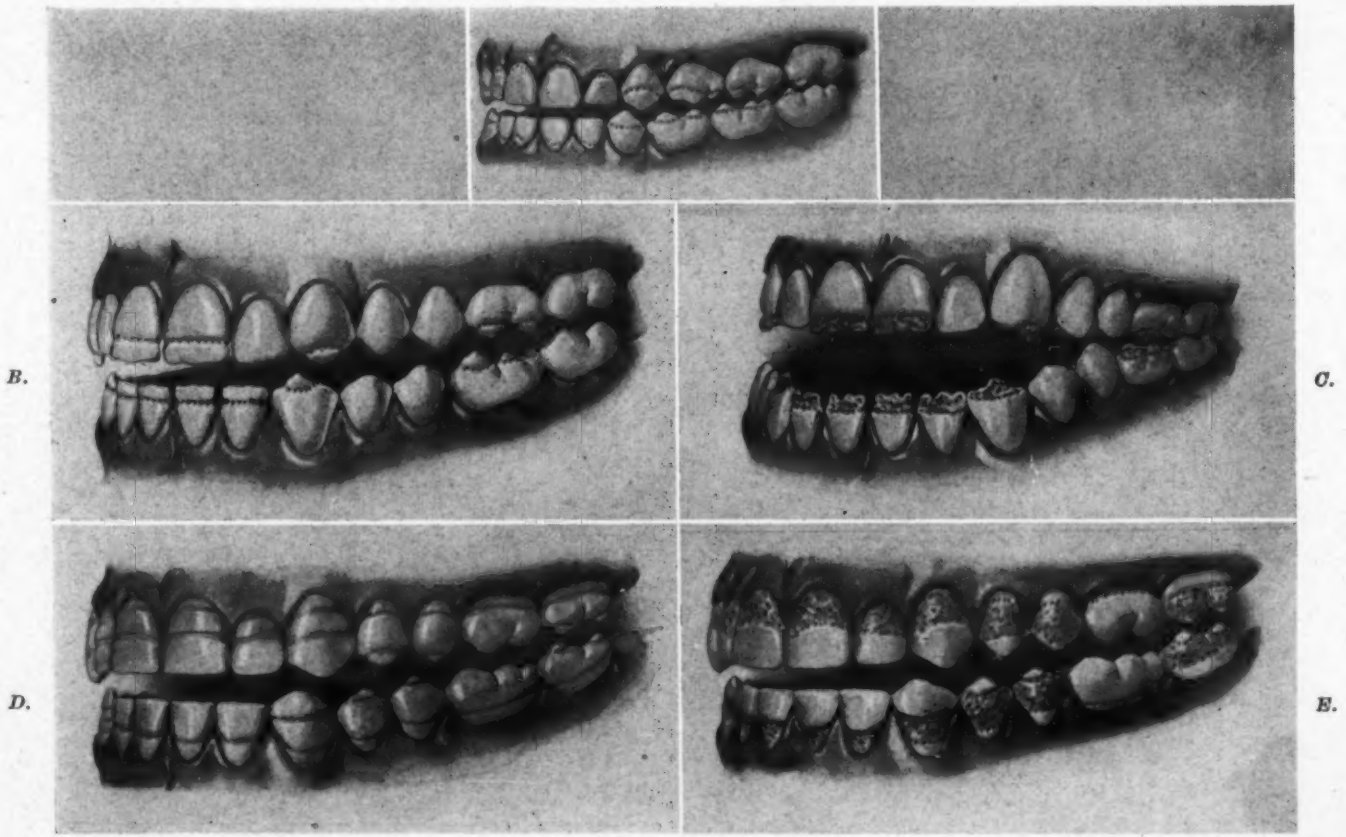

Fig. 6.—Characteristic types of enamel hypoplasia. Hypoplasia of the infancy ring (acute) or infancy period (chronic) may or may not affect the tip of the upper lateral incisor. *A*, Neonatal hypoplasia. *B*, Acute hypoplasia of the infancy ring (10 months). *C*, Chronic hypoplasia of the infancy period (birth to 10 months). *D*, Acute hypoplasia of the early and later childhood rings (2½ and 4½ years). *E*, Chronic hypoplasia of the childhood period (2½ to 4½ years). (From Massler and Schour: Atlas of the Mouth, American Dental Association.)

"Rickets is dependent on bone growth. The disease in its manifest form cannot occur without growth of the skeleton, because lime salts cannot fail to deposit unless new-formed bone tissue exists for them to fail to deposit in. Rickets is present or absent according to the relationship between rate of lime-salt deposition and rate of bone growth. If the latter just corresponds to the former, calcification of the bone is complete; if the latter exceeds the former, osteoid tissue makes its appearance and rickets develops."

Mellanby[1a] expressed the opinion that the decrease in the quality of the calcification of the teeth during infancy is due to an increased rate of growth of enamel and dentin during the latter part of the first year. This is doubtful, since the most rapid rate of growth of enamel and dentin, as well as the best calcification, occurs prenatally. The rate of formation of enamel and dentin follows definite laws of growth gradients, whereas the calcification pattern disregards these rates and gradients of growth.

Discussion.—That the calcification of the dental tissue is normally relatively poorer during the first ten months of life and that 60 per cent of all dental hypoplastic defects occur within this period (chronic hypoplasia of infancy) and usually cease suddenly thereafter, are significant clinical observations for which an explanation should be sought by the pediatrician. The period of infancy is a continuation of the postnatal adjustments in the circulatory system, the gastrointestinal tract and, in fact, in all metabolic processes which are initiated at birth and carried through the neonatal period. The change from the parasitic intrauterine existence to the more independent and traumatic postnatal type of life is not completed for some time after birth. The major adjustments must be made quickly during the neonatal period but many minor adjustments continue for a long time thereafter. In addition, the rapid prenatal rate of growth continues (with but a temporary interruption at birth) to the tenth month of age. However, the food supply is changed from a passive, prenatal, placental transfer to a somewhat less efficient postnatal gastrointestinal digestion and absorption. For these reasons—and others—the first ten months of life is a period during which the metabolism and the cellular activities are highly susceptible to constitutional disturbances.

Scammon[23] pointed out:

"From the standpoint of growth, practically all of infancy may be regarded as a transition period, in which the organs and structures of the body are departing, one by one, from the simple style of increment characteristic of the fetus to the diverse and complex modes of growth that obtain in childhood."

The fact that the enamel and dentin formed during the first three months of life show relatively better calcification than those formed during the subsequent six months suggests that the fetal mineral reserves have not yet been exhausted. A correlative fact well known to those practicing pediatrics is the relative freedom from rachitic manifestations during the first three months of life in spite of a vitamin D deficiency present since birth.

Infancy Ring

Findings.—At the age of 10 months (the absolute time and the position of the ring vary slightly) a sharply accentuated incremental line can constantly be observed in the enamel and the dentin of both the deciduous and the permanent teeth forming and calcifying at that time (Figs. 2 and 4). Because of the period at which it appears, we have termed this accentuated incremental line the infancy ring. Since the upper lateral incisor begins its formation at about the age of 10 months, the line is seen only in histologic sections taken immediately over the growth center.

In the deciduous teeth the line was observed only in the dentin, since formation of the enamel of these teeth had already been completed at this time (Fig. 2). In these teeth it appeared in about 75 per cent of the sections as a hypocalcified line at the junction between two characteristic zones of calcification. In the enamel of the permanent teeth forming and calcifying at the age of 10 months (anterior teeth and first permanent molar), the infancy ring was observed in over 90 per cent of the sections as an accentuated band of Retzius that also demarcated two zones of calcification. The corresponding line in the dentin of the permanent teeth was much less prominent and was discernible in only about 50 per cent of the sections, although the different zones of calcification were readily apparent. In the teeth in which it was not apparent the infancy ring was considered to be at the junction of the two zones.

Hypoplastic defects: In chronic hypoplasia of the infancy period the infancy ring usually marks the limit of the chronic hypoplastic zone. It also represents the severest effect of the chronic hypoplasia. It can be recognized grossly by its marked accentuation in the form of deep pittings and represents the most intense manifestation of the metabolic disturbance, as well as its abrupt termination.

About 15 per cent of the total of hypoplastic defects were acute and confined to the chronologic level of the infancy ring (acute hypoplasia of the infancy ring). They were usually of the pitted variety (Sarnat and Schour[20]). Such defects affect the first permanent molars and the anterior teeth, except the upper lateral incisors (Fig. 6).

Review of Literature.—In describing certain regularly recurring and prominently accentuated lines of Retzius, which he stated might be caused by the decreased amount of sunshine during the winter months, Swanson[2a] stated:

"Broad incremental lines sometimes evident in the tips of the central incisors and in deposits of enamel of first molars which represent the accretions of about 1 year (*corrected to about ten months**) of age are possibly caused by nutritional disturbances associated with weaning."

Berten,[24] in 1895, and Asper,[25] in 1916, described deficiencies in the formation and calcification of teeth at about the same chronologic level which they

*Italics mine.

also associated with the process of weaning. This interpretation is questionable in the light of present-day knowledge. In modern times, weaning is a gradual process which often begins at birth. Yet the infancy ring is just as prominent today as in 1896, when the experience of weaning was more traumatic. In addition, the actual time of weaning varied considerably in our material, yet the infancy ring showed a high degree of constancy in the time at which it occurred.

Review of Literature.—It is likely that the infancy ring is a result of a profound adjustment in the internal conditions of the developing child at about ten months of age. In 1915 Robertson[26] reported the existence of a "critical period" in the latter half of the first year of the extrauterine development of man. The existence of this critical period was established on the basis of the observations of MacGregor[27] and has since been confirmed by the work of Thompson.[28] These investigators reported a decline in the growth curve at 6 to 12 months of age. The infant tends to become relatively underweight at the age of 7 to 10 months. In addition, it was found that the incidence of certain diseases of infancy, such as diphtheria, cerebrospinal meningitis, measles, whooping cough, and scarlet fever, was maximal between the ages of 6 and 9 months.

Since the critical period falls at the link between two growth cycles, Robertson expressed the belief that it was due to a change from the metabolism of the first growth cycle to that of the second.

Discussion.—The infancy ring seems to demarcate the period of postnasal adjustments from the relatively more independent and robust existence of early childhood and thus may be compared with the neonatal ring as a critical point in the constitutional pattern of the developing child. Like the neonatal period, the tenth month is a period during which metabolic and cellular activities are acutely susceptible to systemic disturbances. This is evidenced by the fact that the infancy ring is normally an accentuated incremental ring. In addition, patients who show a severe and acute hypoplastic defect at the level of the tenth month give no history of illness at that time.

While it is not difficult to explain the existence of the neonatal ring, the discovery of the possible underlying cause of the infancy ring presents a challenge to the pediatrician and the investigator of child growth which has not yet been met. It would not be surprising to find a corresponding ring in the long bones of normal infants of about one year of age.

Calcification During Early Childhood

The period between the end of the first year and the end of the second year of life was considered to be the period of early childhood. Figs. 2 and 4 show the teeth and the levels which form during this period. Note that during this time the gingival third of the anterior teeth and the first permanent molars are forming. The premolars and second molars are just beginning their formation at the end of this period.

Findings.—The dental tissues show, in general, calcification which is more homogeneous than that occurring during the infancy period but less homogeneous than that of the tissues calcified before birth.

The enamel formed during this time over the dentin cusps in the premolars and second molars is white, translucent, and free from striae of Retzius. It has been called cuspal enamel[29] and compares in density with the rest of the enamel as mantle dentin compares with circumpulpal dentin. The cuspal enamel and the mantle dentin are constant findings and are not related to the chronologic development of the child.

The dentin forming and calcifying during this period shows a decrease in the incidence of interglobular dentin as compared with that in the infancy zone, but marbled or dappled dentin is common.

Hypoplasia of the enamel: Recovery from hypoplastic defects of the infancy period is complete and abrupt at the beginning of the early childhood period (Fig. 6). Chronic hypoplastic defects of the early childhood period are relatively rare.

Discussion.—During early childhood, in contrast to infancy, the metabolic and cellular processes are relatively immune to systemic disturbances. Many patients who show no clinical evidence of improvement show a remarkable recovery in the calcification of the dental tissues as well as a relative immunity of the epithelial, enamel-forming cells. The recovery from the marked disturbance in the calcification and the formation of the teeth during infancy is abrupt and definite. The calcification of the teeth and the cells responsible for enamel formation, which were so highly sensitive during infancy even to subclinical systemic disturbances, now appear immune to disturbances which are clinically manifest.

Early Childhood Ring

Findings.—A sharply accentuated ring can be observed in the permanent premolars and second molars at the level of the enamel and dentin, forming at about 2½ to 3 years of age (Fig. 4). The line is readily observed in the enamel, being present in about 75 per cent of the sections, but it is not so easily seen in the dentin, where it was observed in only about 25 per cent of the sections. It is also not so accentuated as the neonatal or infancy rings. Its histologic appearance in the enamel, and in the dentin also, is similar to that of the infancy ring. It probably represents a line of arrested growth.

Hypoplastic defects of the early childhood ring: About 5 to 10 per cent of all of the hypoplastic defects were confined to the level of the early childhood ring. These were acute and were generally smooth, whereas the acute hypoplastic defects of the infancy ring were generally deeply pitted (Fig. 6).

Discussion.—The early childhood ring represents a chronologic level during which the metabolic and cellular activities were acutely susceptible to systemic disturbances. This is evidenced by the fact that the early childhood ring is normally more prominently accentuated than the other incremental rings formed during this period and by the increased incidence of hypoplastic defects in the enamel at this particular chronologic level. The reason for this susceptibility is not known.

Calcification During Later Childhood

The period from the beginning of the third year to the end of the fifth year is usually designated as later childhood. The levels of the teeth forming and calcifying during this time are shown in Figs. 4 and 5.

Findings.—In general, the calcification after the third year is less homogeneous than that occurring during the early childhood period but better than that occurring during the infancy period. The enamel of the permanent teeth contains a distinctly greater number of incremental lines which are characteristically more heavily pigmented than those lines which occur at an earlier chronologic level. The dentin tends to exhibit a greater degree of interglobularity than that formed during the early childhood period but a lesser degree than that formed during the infancy period.

Hypoplasia of the enamel: About 5 to 10 per cent of the total number of hypoplastic defects occur during this period and are of the chronic or multiple acute type. Clinically, they are usually seen only in the cuspids, the premolars, and the second molars (Fig. 6). In the incisor teeth the defects, when present, are confined to the area close to the cementoenamel junction, and in children they are generally covered by the gingival tissue and are therefore easily overlooked. The hypoplastic defects occurring during later childhood are generally of the smooth variety and give to the enamel a wavy appearance rather than the pitted appearance caused by those occurring during the infancy period. For this reason, too, they are often overlooked.

Discussion.—The decrease in the quality of the calcification and the higher incidence of hypoplastic defects in the enamel during the period from the third to the fifth year indicate an increased susceptibility of the dental tissues to metabolic and cellular disturbances. This susceptibility, however, is much less than during infancy, though greater than during early childhood.

Sarnat and Schour[20] have shown that there is no correlation between the exanthems which occur during later childhood and the hypoplastic defects which occur during infancy. The factors involved in the causation of the hypoplasia of the infancy period are still unknown. The increase in the incidence of hypoplastic defects in the enamel may, however, be related to the exanthematous diseases which occur during this period. This problem merits further investigation.

Later Childhood Ring

Findings.—A ring is frequently present in the enamel of the premalors and second molars near the level of the cementoenamel junction and in the dentin of the roots of the other permanent teeth at a level that corresponds to the fifth year of life (Figs. 4 and 5). The ring was observed in the enamel of 80 per cent of the sections; it has the typical accentuation and appearance of a line of arrested growth. In the dentin the line is not markedly accentuated and was observed in only 60 per cent of the sections studied. Any accentuated incremental ring is as a rule more easily recognized in the enamel than in the dentin. The later childhood ring both in the enamel and in the dentin is less prominently accentuated than is the infancy or the neonatal ring.

Hypoplastic defects of the later childhood ring: As in the case of the infancy ring, the later childhood ring generally marks the termination of the chronic hypoplastic defects occurring during the preceding period. About 5 per cent of the total number of hypoplastic defects are of the acute type and are confined to the level of the later childhood ring. They usually are of the smooth variety and can be observed only in the gingival third of the cuspids, premolars and second molars (Sarnat and Schour[20]). Because these defects are smooth and represent a "wave" in the enamel, they often are overlooked.

Discussion.—The later childhood ring, like the early childhood ring, represents a chronologic level of acute susceptibility to systemic disturbance, as evidenced by the fact that it is normally accentuated and that about 5 per cent of all hypoplastic defects occur at this level. The reason for this susceptibility is not known.

Calcification During the Grade-School Age

The grade-school age begins with the sixth year and continues until puberty. Enamel formation is being completed in the premolars and second molars but is just beginning in the third molars. The records during this period are therefore written in the dentin of the roots of the premolars and second molars and in the enamel of the third molars. Enamel formation in the third molars and root formation in the premolars are completed between the twelfth and the fifteenth year.

The beginning and the rate of formation of the third molar are inconstant. Its root formation is unpredictable. The result of any attempt to date the incremental pattern, therefore, can be only an approximation. Therefore, the dating of rings in the third molar will not be attempted until we have studied further the chronologic development of this tooth.

As previously indicated, rings are relatively more difficult to observe in the dentin than in the enamel. Therefore, the recording of rings in the teeth is virtually ended by the tenth year of life. However, the quality of the calcification from the eighth to the thirteenth year can be estimated in the root dentin of the premolars and second molars and the enamel and the dentin of the crown of the third molars.

Findings.—The calcification of the dentin from the sixth to the thirteenth year is fairly homogeneous. Dappled dentin is common, but markedly interglobular dentin is infrequent. The calcification, therefore, is better than that occurring during infancy and is comparable to that of the early childhood period.

The enamel of the third molars is well calcified, although striae of Retzius are common, except within the area of cuspal enamel.

Hypoplasia of the enamel: Hypoplasia of the enamel, which could affect only the third molars during this period, was virtually absent in our material.

Discussion.—The grade-school age represents a period of improved calcification and decreased susceptibility to hypoplasia of the enamel.

RÉSUMÉ OF THE HISTOLOGIC CHARACTERISTICS OF THE ENAMEL AND DENTIN FORMED AND CALCIFIED DURING THE DIFFERENT DEVELOPMENTAL PERIODS OF THE CHILD

Prenatal Period (4 months in utero to birth).—The formation and calcification of the prenatal tissues are normally good and are usually unaffected even in cases of disturbed metabolism of the mother. Only very severe deficiencies in the mother affect the tissues calcified before birth.

Neonatal Ring (birth to 2 weeks of age).—The neonatal ring is an arrest line which is probably the result of the change from the intrauterine to the extrauterine physiology. Any trauma associated with birth tends to accentuate the neonatal ring.

Infancy Period (2 weeks to 10 months of age).—Calcification is normally not homogeneous during infancy. It is easily affected even by subclinical disturbances in the metabolism of the growing infant. Hypoplastic defects in the enamel frequently result from disturbances which are not apparent clinically. This period is critical for the formation and calcification of the teeth.

Infancy Ring (about 10 months of age).—The infancy ring is an arrest line which normally results from unknown factors occurring at about ten months of age. It represents a critical period in which the formation and calcification of the enamel and dentin are acutely susceptible to subclinical disturbances in the metabolism of the growing child. The susceptibility is even greater than during the infancy period, and the response in the enamel and dentin is even more manifest.

Early Childhood Period (about 10 months to 2½ years of age).—The formation and calcification of the enamel and dentin during this period are good, although not so good as during the prenatal period. It is a period of relative immunity to disturbances in the formation and calcification of the enamel and dentin, since disturbances in the metabolism of the growing child, which are apparently clinical, usually fail to produce enamel hypoplasias and cause only a relatively slight disturbance in calcification. The recovery from hypocalcification and hypoplasia occurring during infancy is abrupt and complete.

Later Childhood Period (about 2½ to 5 years of age).—The calcification of the enamel and dentin is normally not so homogeneous as during early childhood, but it is better than during infancy. Disturbances in formation and calcification do occur in response to disturbances in the metabolism of the growing child which are clinically manifest.

Grade School Period (about 6 to 10 years of age).—This period, like the early childhood period, is characterized by relatively good calcification and immunity to disturbances in enamel formation.

Early and Later Childhood Rings (about 2½ and about 5 years of age).—The early and the later childhood rings occur at the beginning and at the end of the later childhood period, just as the neonatal and the infancy rings demarcate the infancy period. They are probably the result of unknown factors operating in the child at about 2½ and at about 5 years of age. They

TABLE III. DEVELOPMENTAL PATTERN OF THE CHILD AND ITS REFLECTION IN THE TEETH

DEVELOPMENTAL PERIODS AND TOOTH RINGS	AGE PERIOD	QUALITY OF CALCIFICATION AND HISTOLOGIC CHARACTERISTICS	DISTRIBUTION OF HYPOPLASTIC DEFECTS IN THE ENAMEL*
Prenatal period	4 to 9 m.i.u.†	Good calcification	Rare
Neonatal ring	Birth to 2 weeks	Dark, distinct arrest line in enamel and dentin	3% (Acute hypoplasia of the neonatal ring)
Infancy period	2 weeks to about 10 months	Period of poorest calcification and greatest susceptibility to chronic hypoplastic defects	60% (Chronic hypoplasia of the infancy period)
Infancy ring	About 10 months	Sharp arrest line in enamel and dentin demarcating infancy from early childhood period. Marks a period of acute susceptibility to hypoplastic defects of the enamel as well as the abrupt termination of chronic hypoplastic defects of infancy	15% (Acute hypoplasia of the infancy ring)
Early childhood period	About 10 months to 2½ years	Recovery in calcification and cessation of hypoplasia abrupt and complete. Calcification better than during infancy but not so good as during prenatal period	Relatively rare
Early childhood ring	About 2½ years	A sharp arrest line in enamel and dentin which demarcates early from later childhood periods. Marks a period of acute susceptibility to enamel hypoplasia	5-10% (Acute hypoplasia of the early childhood ring)
Later childhood period	About 2½ to 5 years	Calcification generally poor but better than during the infancy period	5-10% (Chronic hypoplasia of the later childhood period)
Later childhood ring	About 5 years	A sharp arrest line in enamel and dentin which demarcates the later childhood from the grade school periods. Marks a period of acute susceptibility to enamel hypoplasia	5% (Acute hypoplasia of the later childhood ring)
Grade school age	About 5 to 10 years	Calcification generally good but variable	Relatively rare

*About 5 per cent of the population suffers from hypoplastic defects. The figures give the distribution of these defects according to age and refer to percentages of the total number of hypoplastic defects observed.

†M.i.u. = months in utero.

represent critical periods, in which the formation and calcification of teeth are acutely susceptible to disturbances in the metabolism of the growing child. The susceptibility is less than during the infancy period or at the tenth month of age but greater than during the later childhood period.

SUMMARY AND CONCLUSIONS

The human being goes through successive developmental epochs, each of which involves a complete, and often traumatic, change in metabolic, cellular,

and other constitutional processes. These are reflected accurately and permanently within the structure of the dental tissues forming and calcifying at that particular time. An analogy is found in the tree, of which the annual rings reflect the vicissitudes and variations in climate experienced during the period of growth. To the pedodontist, the pediatrician, and others dealing with children, the quality of the dental tissues should serve as a valuable index of the prenatal care of the mother and of the feeding of the child during infancy and childhood.

Over 1,000 human deciduous and permanent teeth were studied in ground and decalcified sections for the purpose of evaluating the degree of calcification of the enamel and the dentin at different ages. The calcification pattern of the teeth, which is indicated in Table III and in Figs. 2 and 4, is a direct reflection of the physiologic characteristics of the developmental periods of the growing child. In addition, a clinical examination of the teeth of children was made, with emphasis on the incidence of acute and chronic hypoplastic defects in the enamel.

The period in which there are the greatest susceptibility to hypoplastic defects in the enamel and the poorest calcification lies between birth and about 10 months of age. Deficiencies in calcification tend to become progressively accentuated at about 10 months of age, when a crisis occurs and the recovery from hypocalcification and hypoplasia is abrupt and complete. Periods of acute susceptibility to metabolic and cellular disturbances (evidenced as acute deficiencies in formation and calcification) occur at the neonatal period (neonatal ring), at the age of 10 months (infancy ring), at about 2½ years (early childhood ring), and at about 5 years (later childhood ring). The recognition of the constitutional basis for the susceptibility to defects in calcification and formation which occur during the infancy period and at the levels of the infancy and the early and later childhood rings awaits further investigation

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THE TREATMENT OF MALOCCLUSION WITH AND WITHOUT THE REMOVAL OF DENTAL UNITS

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IN THE treatment of malocclusion the orthodontist strives for certain objectives. Primarily he is concerned with the stability of the denture after all retaining appliances have been removed. Will the teeth remain relatively in their new positions? Will they function normally? Will the investing tissues remain healthy? Second, he is concerned with facial esthetics, with emphasis on the dentofacial area. Will the balance, harmony, and beauty of the facial features be enhanced? Or, if not enhanced, will they be maintained and not distorted?

In order to reach these objectives it is imperative that we should be able to interpret correctly "the line of occlusion," as given to us by Dr. Edward H. Angle. According to Tweed,[1] six fundamental requirements must be met if normal occlusion is to be the end result of orthodontic treatment. These requirements are:

1. There must be a full complement of teeth, and each tooth must be made to occupy its normal position.
2. There must be normal cusp and occlusal relationships.
3. There must be normal axial inclinations of all the teeth.
4. There must be normal relationships of the teeth to their respective jawbones.
5. There must be normal relationship of the jawbones to each other and to the skull.
6. Normal function of all the associated parts must be established.

Those of us who have practiced the philosophy of the full complement of teeth have too often discovered that while we have met the first two requirements, we have failed to meet the other four. In sum, we have retained the full complement of teeth and gained normal cusp and occlusal relationships and arch form, but in the majority of instances have failed to gain normal axial inclinations, normal relations of teeth to basal structures, and normal function of associated structures. The result has been nonpermanency of tooth positions and loss of balance, harmony, and beauty of the face. We have found that in the majority of our treated cases there was either relapse, not of one tooth but of many, or complete collapse of the entire denture. This record is not one in which we can take pride.

As a result of his clinical research, Tweed has stated that, while we should always strive to meet all six requirements of normal occlusion, we are sometimes confronted with conditions that preclude the possibility of maintaining a full

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complement of teeth, and that at times we must remove dental units in order to meet the other five requirements.

This conclusion has been justified by the works of Broadbent and Brodie on the growth of the osseous structures of the face. As a result of his work on facial growth, Broadbent has stated that misadventures in health during the growth and development period affect the osseous growth, and that a loss in bone growth in one period is never regained or made up at a future date. Brodie has stated that it is impossible to make basal bones grow, and this has become a truism in the field of orthodontics.

Thus we find ourselves in a position where we must choose when to retain a full complement of teeth and when to compromise and remove dental units. This choice is not always simple, though there are certain signs and symptoms that are readily observable and may be utilized by all practicing orthodontists. It is with some of these diagnostic signs that this paper is concerned.

Fig. 1.—Normal profile of face, and profile model of mandibular incisors showing upright position of incisors.

If we examine a number of normal dentures in which no orthodontic therapy has been instituted, we note certain characteristics common to all of them. Not only are there normal cuspal and occlusal relationships; but also the teeth are more or less upright on basal bone and there is a certain balance, harmony, and beauty of facial features in accordance with type (Fig. 1). There may be some slight discrepancies in the alignment of the incisors, but nevertheless the incisors remain upright over basal bone. Margolis, Broadbent, and Brodie have shown that the angulation of the lower incisors is from 85 to 95° when measured from the mandibular plane. Hence the justification of Tweed's minus five to plus five position of the mandibular incisors.

If we have this picture ever present in the back of our minds and can accept the upright position of the mandibular incisors on basal bone as representing Nature's plan in building a normal denture, it becomes a relatively simple matter to determine in most cases when and when not to remove dental units in the treatment of malocclusion.

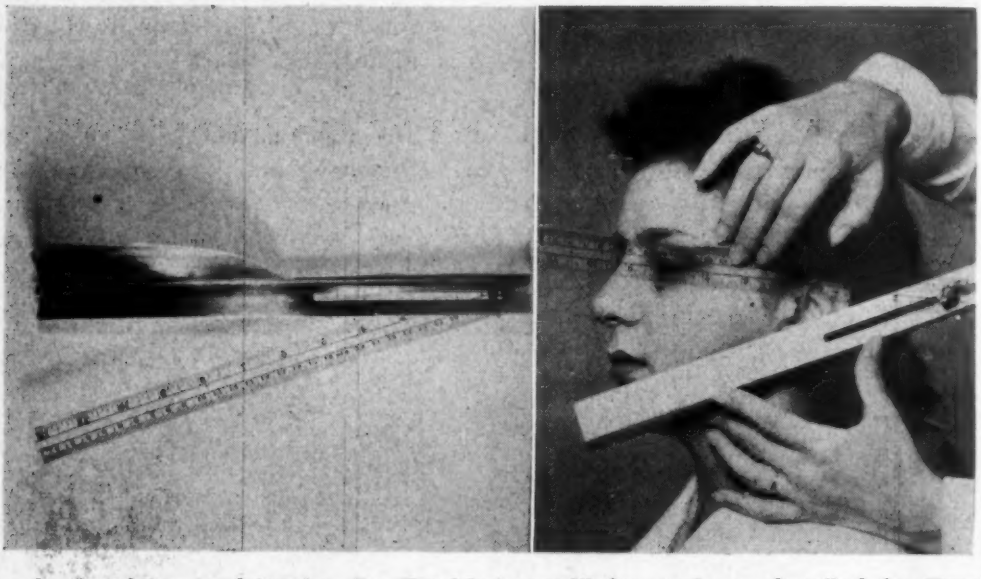

Fig. 2.—Angulator to determine the Frankfort mandibular angle as described by Tweed. Dr. B. L. Herzberg designed the angulator.

In the light of our newer knowledge it is possible to analyze the face and interpret these findings, and thus to determine what procedure to follow. Tweed,[2] in a recent presentation on the Frankfort-mandibular angle and its significance in prognosis, makes the following statement:

The Frankfort-mandibular angle is formed by the intersection of the Frankfort horizontal plane extended distally beyond porion and the plane of the lower border of the mandible with the teeth in occlusion. The Frankfort plane is that plane passing through both orbital points (the inferior border of the orbit) and through porion on the left side of the face.

When the Frankfort-mandibular angle is from 15 to 25° the intersection of the two planes takes place at a point from 4 to 8 or more inches distal to porion. Most normal occlusions are found within this range. Also within this range are found the normal variations of minus 5 to plus 5 inclinations of the lower incisors as described by Tweed. [Tweed thinks that 60 to 70 per cent of all malocclusions fall in this group and that the prognosis is excellent. One half of all malocclusions falling within this group will require extractions in treatment.]

When the Frankfort-mandibular angle is from 25 to 30°, prognosis is good, and in treatment a larger percentage of extractions will be required.

When the Frankfort-mandibular angle is from 30 to 35°, prognosis is doubtful, and when the angle is over 35°, prognosis is bad. From 25 to 35° virtually all cases require removal of teeth in treatment. When the Frankfort-mandibular angle is 30° or upwards, the minus 5 to plus 5 relationship of the mandibular incisors to base does not apply.

As the Frankfort-mandibular angle increases from 35 to 55°, prognosis is nil. In most instances removal of teeth in this range will complicate matters and detract from rather than enhance facial esthetics."

While it remains for many of us to test Tweed's conclusions further before applying them universally, I, as well as others who are followers of the Tweed philosophy and technique, have found the method very useful in most cases in determining when and when not to remove dental units. Dr. B. L. Herzberg of Chicago has recently devised a very useful instrument that simplifies determining the Frankfort-mandibular angle in the living subject (Fig. 2).

Perhaps the best method of describing some of the diagnostic signs and symptoms that help to determine when to maintain a full complement of teeth, and when not to, is to present a series of cases that illustrate how it is done.

CASE 1.—This is a Class II, Division 1 case (Fig. 3). The profile models show the mandibular incisors at an abnormal angle of inclination. They are in protrusion. If we examine the occlusal aspect we find that the arches are well developed and that there is quite a bit of space between the incisors. It is a simple matter to upright these incisors, prepare anchorage in the mandibular arch, use Class II mechanics, and correct arch and jaw relationships. The FMA* is 21°, and the prognosis is good. The facial esthetics have been improved and have remained so. The removal of dental units is contraindicated in such cases.

CASE 2.—This is a Class I malocclusion belonging to the so-called bimaxillary protrusion category (Fig. 4). If we examine the profile models, we note the same sort of protrusion of the mandibular incisors as in Case 1. However, if we examine the occlusal aspect, we note an entirely different picture. Instead of spaces we have a slight crowding of the incisors. The dentofacial area is in protrusion, though the FMA is only 22°, denoting a favorable prognosis.

In order to correct the axial inclination of the mandibular incisors and iron out the alignment, we must either move the entire denture distally or expand the arches to such a degree that the posterior teeth would be moved off their base. Since distal movement of the entire denture is at best a questionable procedure and too much expansion of the arches does not ensure permanency of tooth position, there seems but one solution, and that is the removal of all four first premolars. It is then a comparatively simple procedure to move the anterior segment distally, correct the axial inclination of the incisors, and enhance the harmony and balance of the face.

This case has been out of retention for fourteen months, and the denture seems stable. The face remains as it was at the end of active treatment.

CASE 3.—This case is another bimaxillary protrusion case, which I reported[3] before the Midwestern Component of the Edward H. Angle Society in October, 1943. At that time I reported that in this type of case the face could not be helped appreciably, owing to the angle of the mandible, and that all that could be accomplished was to reduce the protrusion and eliminate the symptoms the patient described when presenting herself for treatment. These symptoms were dryness of the lips and mouth, difficulty in closing the lips, and difficulty in pronouncing certain syllables, especially when singing (Fig. 5).

The profile models show a decided protrusion of both mandibular and maxillary incisors. There is also some crowding of the mandibular incisors, as shown in the occlusal models. The FMA is 31°, which, according to Tweed, makes prognosis doubtful. To correct the axial inclination of the incisors without overexpansion of the arches, a rather hazardous procedure, it was necessary to remove all four first premolars. When the anterior segment was moved distally, the difficulty the patient had in closing her lips disappeared, and the other symptoms also disappeared.

There is very little change in the facial esthetics, but the features have not been distorted The models on the right of Fig. 5 show the case out of retention for twelve months.

*The Frankfort-mandibular angle is thus designated in the remainder of the paper.

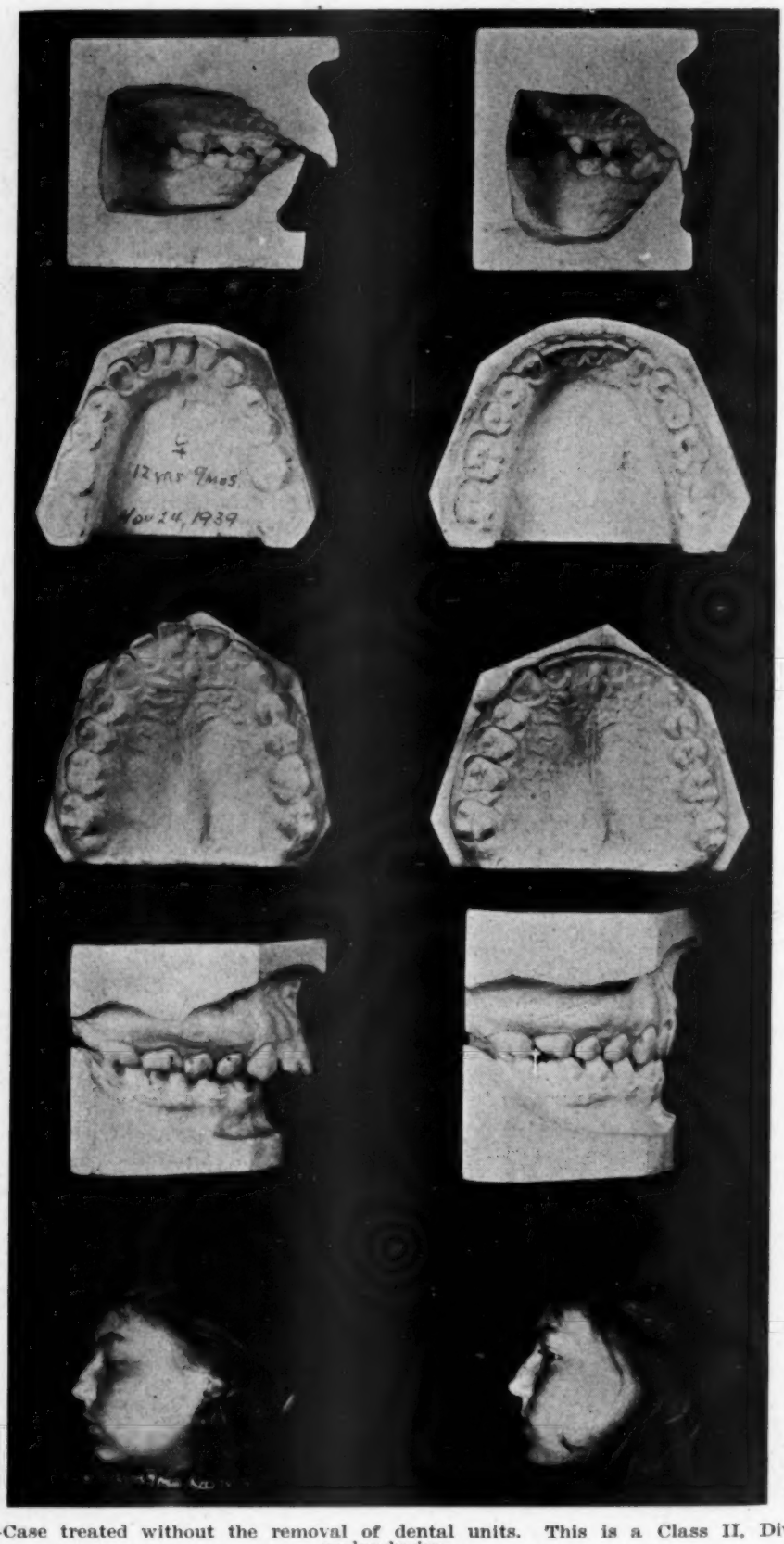

Fig. 3.—Case treated without the removal of dental units. This is a Class II, Division 1 malocclusion.

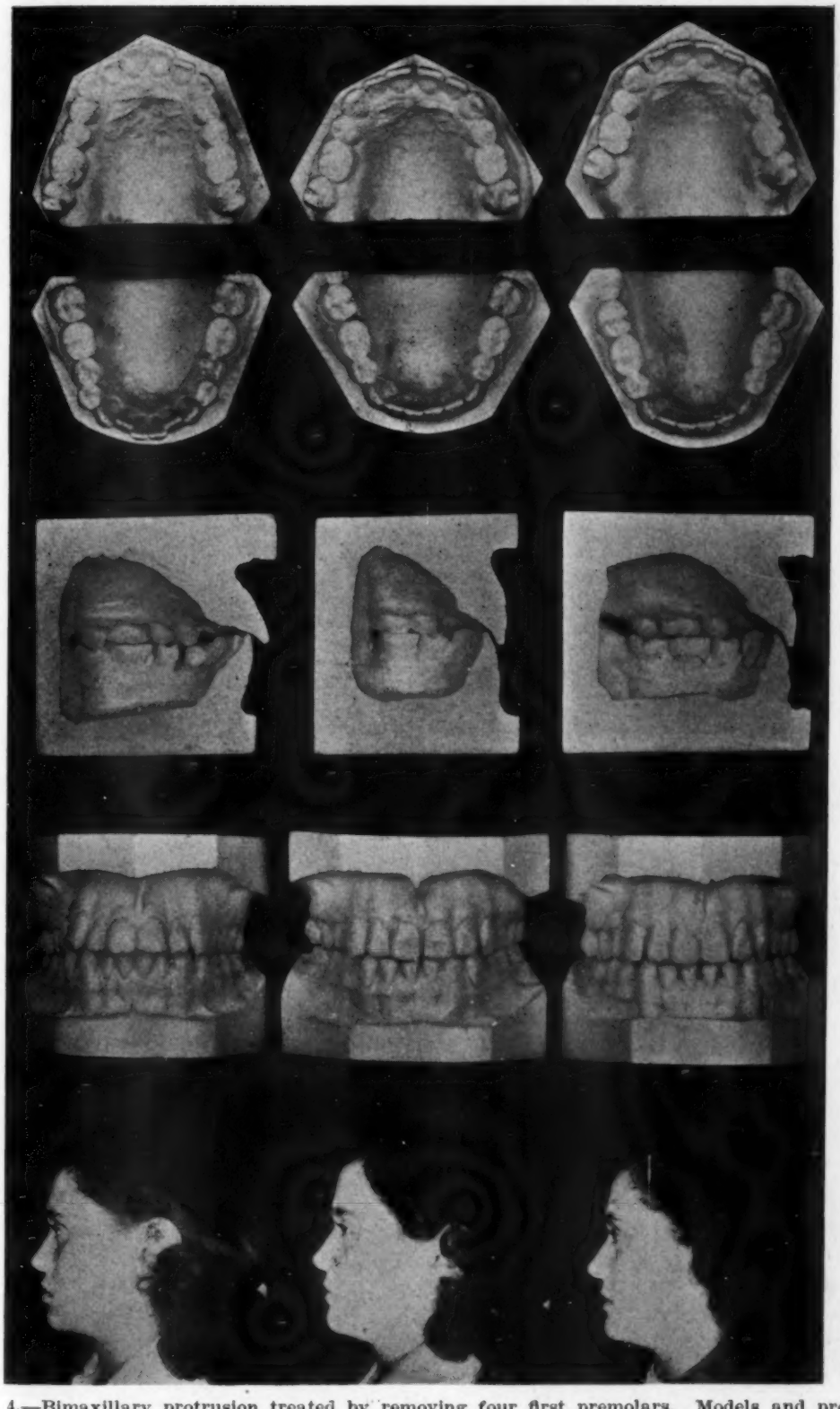

Fig. 4.—Bimaxillary protrusion treated by removing four first premolars. Models and profile to the right taken one year after all retention had been removed.

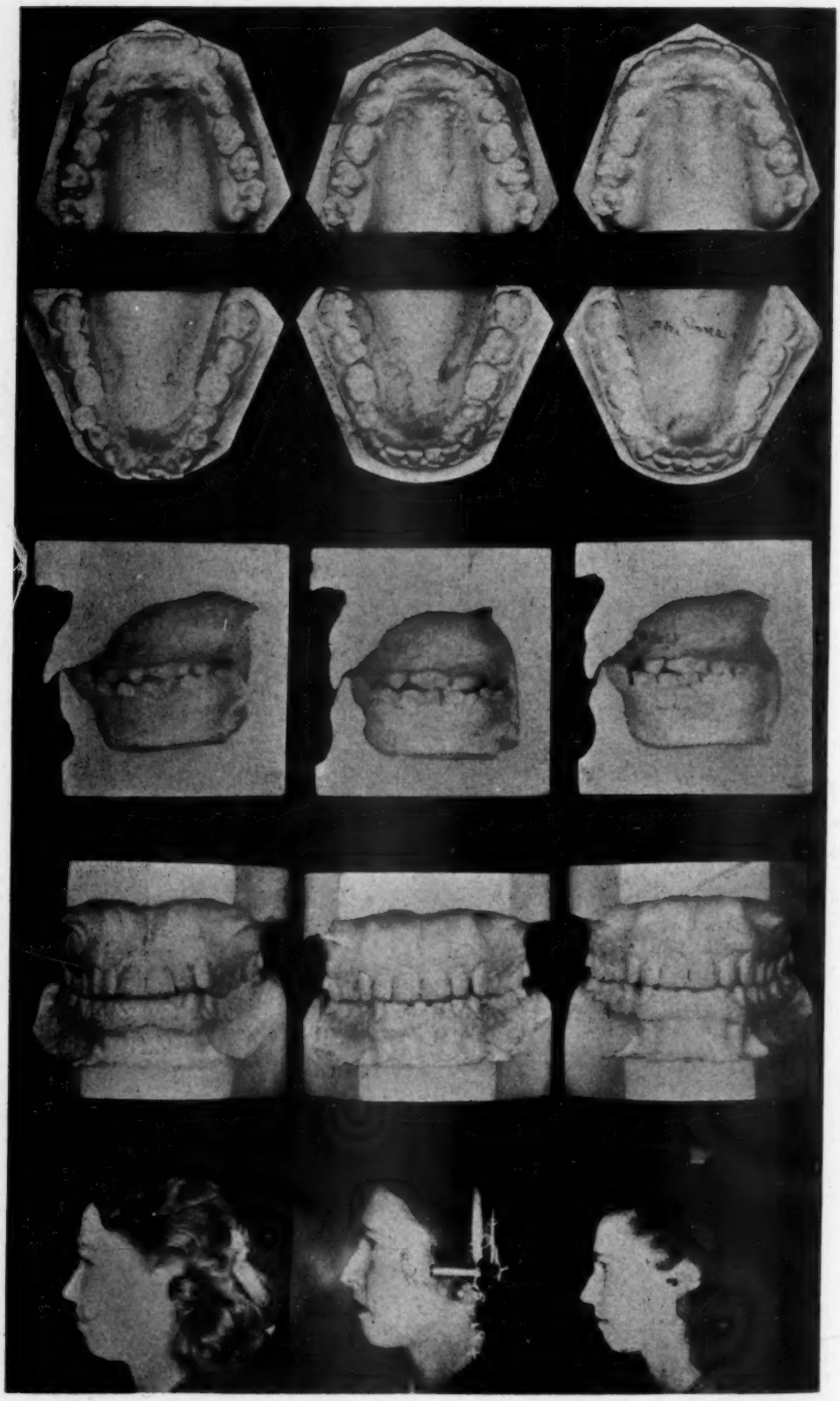

Fig. 5.—Bimaxillary protrusion treated by removal of four first premolars. Models and face profile to the right were taken twelve months after all retention had been removed.

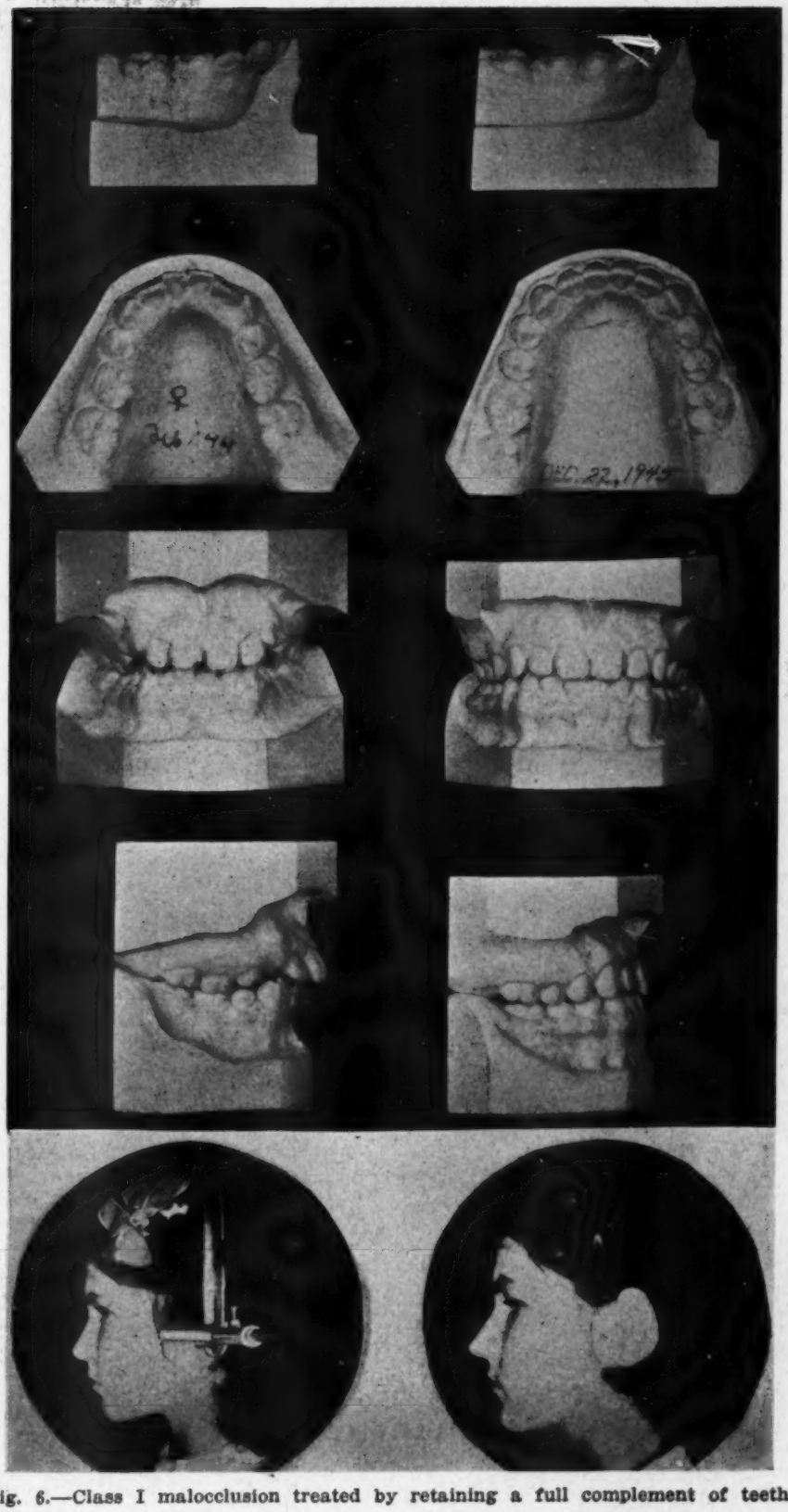

Fig. 6.—Class I malocclusion treated by retaining a full complement of teeth.

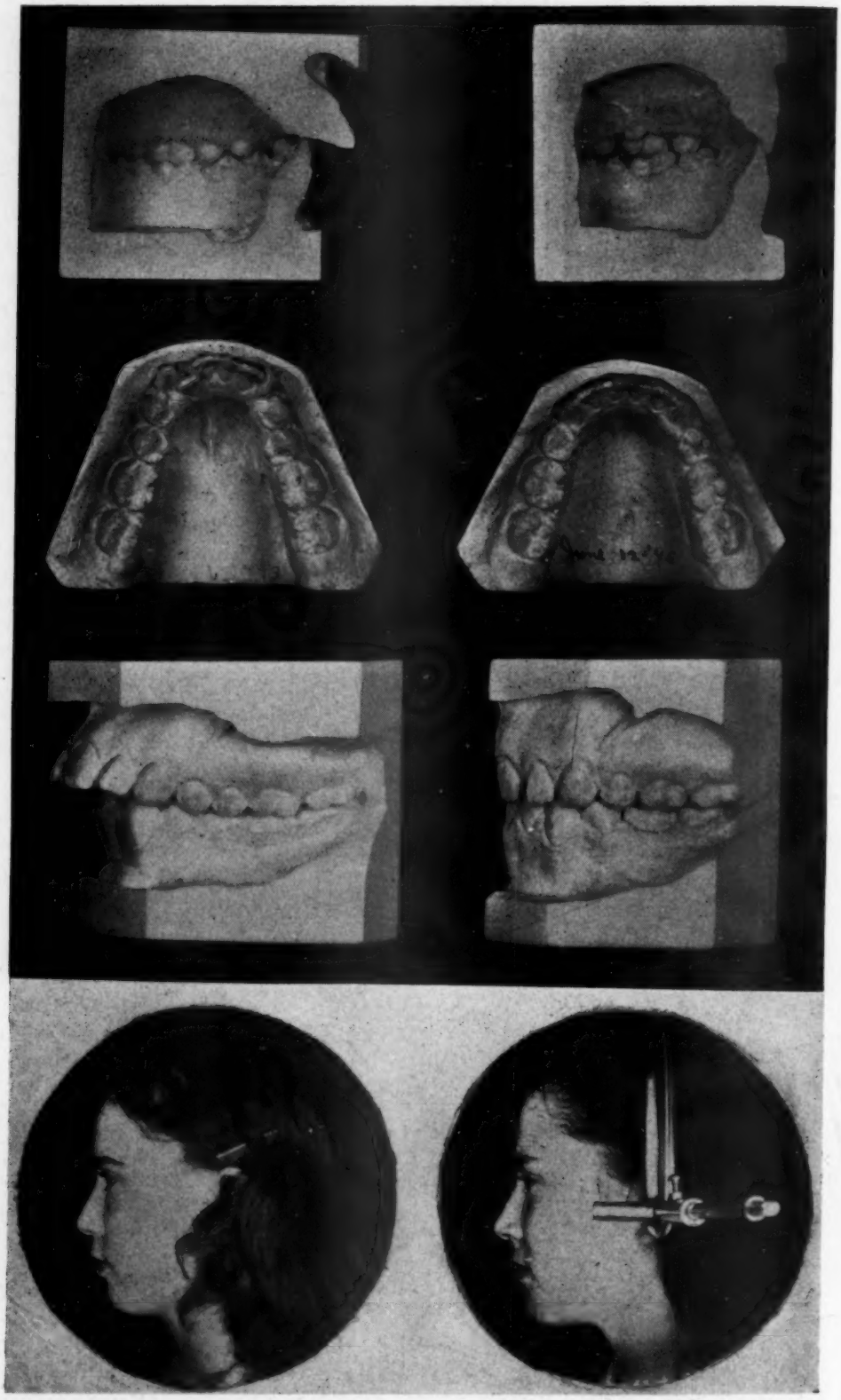

Fig. 7.—Bimaxillary protrusion treated by removing four first premolars. Note the aggravated irregularity in the incisors and narrow arches. Face improvement very marked.

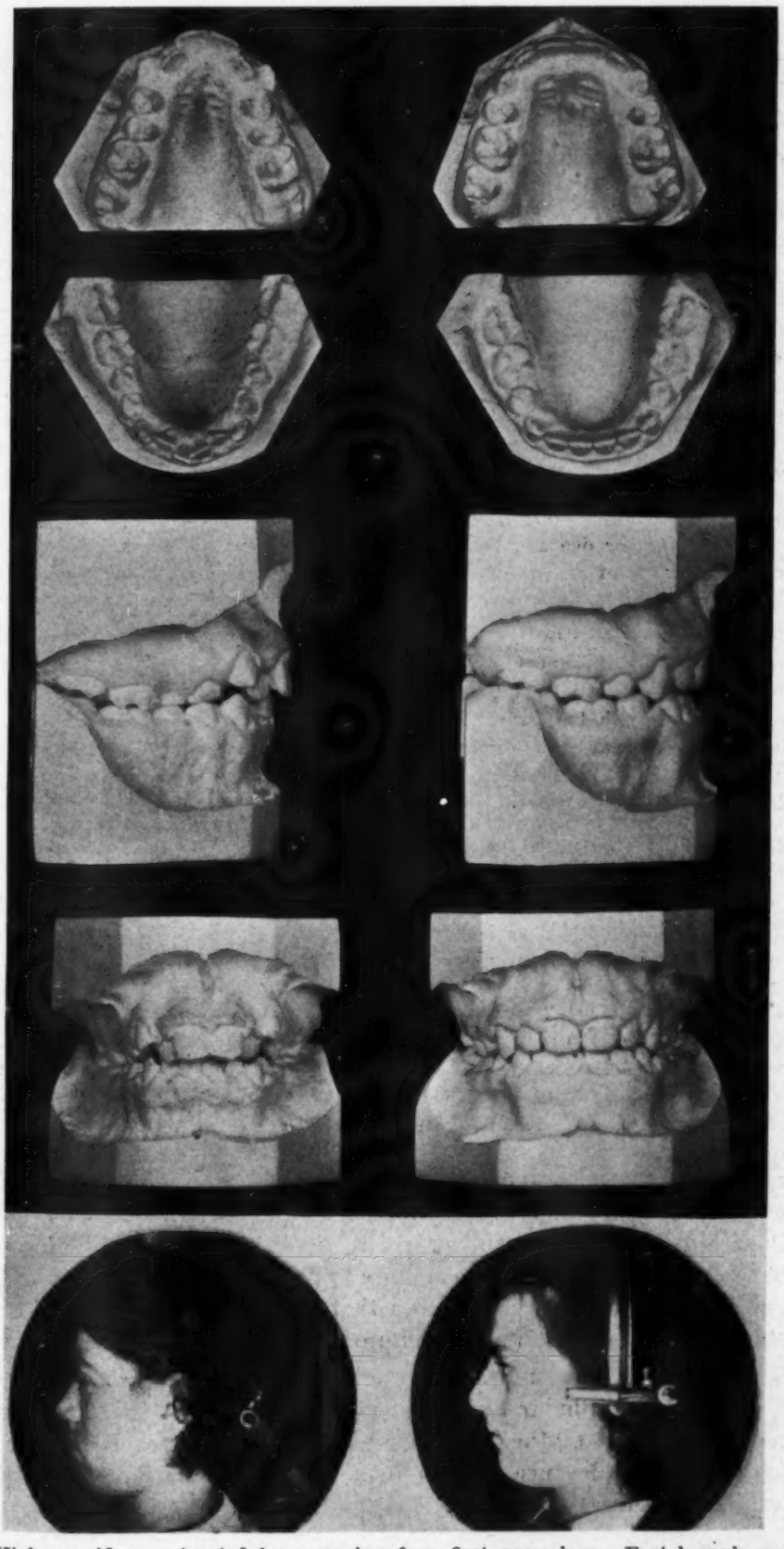

Fig. 8.—High cuspid case treated by removing four first premolars. Facial angle not good.

The denture is a stable one, and should remain so. To have retained a full complement of teeth in this type of case—a rather common type—would have invited sure failure and a collapse of the entire denture.

CASE 4.—In this case (Fig. 6) there is some crowding of the mandibular incisors, but the arches seem well developed. The profile models show the incisors fairly well upright over basal bone. While there is some break in the alignment of the mandibular incisors, it is not sufficient to warrant the removal of dental units in treatment. It is my opinion that while this case may be called a borderline one, it is always well to treat it first by retaining a full complement of teeth. Some lateral expansion is necessary in the treatment of such a case, though not very much. There is usually some slight relapse in the mandibular incisor area, but this is not a serious matter. The FMA is 21°, and thus the prognosis, according to Tweed, is favorable. The active treatment period covered twelve months. The denture was then retained with a Kesling Positioner for three months. The patient is now wearing an upper Hawley plate at night, and though no lower plate is being worn the mandibular incisors are staying in place.

CASE 5.—This case (Fig. 7) is a rather common type in all orthodontic practices. There is a definite protrusion of the denture, the arches are narrow, and the break in the continuity of the mandibular arch is of such proportions that no correction could be made without sacrificing dental units.

All such cases require extraction of premolars, though the FMA, which is 22°, is very good. To retain a full complement of teeth in this type of case would be to invite downright failure. The correction of this case after the removal of all first premolars took sixteen months, after which the patient wore a Kesling Positioner for six months. She is now wearing an upper Hawley plate only, at night, and it seems quite certain that the denture will remain stable. The improvement in the facial outline is most satisfactory, both to the orthodontist and to the patient.

CASE 6.—This case (Fig. 8) represents a type in which it is fairly simple to decide what technique of treatment to follow. In this high cuspid type there is usually a forward migration of the posterior segments of one or both arches, with no room for the eruption of the maxillary cuspids. In this particular case the FMA, which is 35°, is bad, as is quite evident from the profile of the face. There should be no question about the advisability of removing all four first premolars, even though very little if any improvement in facial esthetics can be gained. Such treatment requires no lateral expansion of the arches, and within a period of twelve months all necessary movement of the teeth can be gained. The patient's smile has been improved, even if her face has not. In such cases the patient wears a Kesling Positioner for two to four months, followed by an upper Hawley plate. This patient is still wearing her upper plate at night, and the denture seems a stable one.

The field of diagnostic signs for and against removal of dental units is, of course, not exhausted in presenting these six cases. However, the majority of cases fall within these types. If you are in doubt about which philosophy of treatment to follow, it is a good bit of advice to proceed with the full complement of teeth philosophy first, unless you find that you must overexpand the arches in order to place the mandibular incisors upright over basal bone.

Also, in Class II cases, unless there is a mandibular incisor angulation of over 95°, or if the mandibular incisors are much crowded, it is better to correct the mesiodistal relation of the arches first, then remove dental units to bring about stability of the denture and an improvement in facial esthetics, if this procedure seems necessary.

It is further advisable to utilize Tweed's Frankfort-mandibular angle to help in determining what methodology to follow. I have found it very useful, especially in borderline cases.

The scope of this paper and the time allotted to it make it impossible to cover the details of treatment. However, if time permits and there are questions that I can answer, I shall consider it a privilege to do so.

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THE FAMILY AND DENTAL DISEASE*

II. Age of Parents and Dental Caries (DMF) Experience in Offspring

Henry Klein, D.D.S., Sc.D.,† Bethesda, Md.

THE first report in this series indicated that there is some association between the amounts of dental caries experienced by husband and wife. Examinations of more than 1,700 married couples revealed that men who have experienced large amounts of dental caries tend to have wives whose caries experience is higher than average, while men relatively free of caries attack have wives whose experience with dental disease is lower than usual.[5] This kind of phenomenon has been noted in other instances: for example, tall persons tend to marry individuals whose heights are taller than average; this characteristic has been discussed by Pearl and termed "assortative mating."[8] In another report it was shown that the amount of dental caries observed in children is related rather closely to the amount of caries attack experienced by their parents.[6] These findings suggest powerful familial influences in susceptibility and resistance to dental decay.

Systematic analysis of the family factors in dental caries should include also some investigation of the relation between age of parents at time of birth of offspring and the amount of caries experienced by the offspring.

It has been clearly demonstrated that age of parents has a direct influence on the viability and well-being of the offspring. Elderton[3] has pointed out that abnormal confinements are least frequent among mothers between 31 and 36 years of age. Dahlberg[1] has shown that the frequency of occurrence of fraternal twins varies with the age of mothers, reaching the highest frequency between 35 and 40 years of age. Duncan,[2] in plotting the weight of newborn infants relative to age of mother, has found that the weight becomes greater as infants of progressively older mothers are studied, reaching a maximum for 25- to 29-year-old mothers and then decreasing as mothers' age advances beyond that age interval. Fasbender[4] reports that the birth weight of children of mature mothers is greater than that of children of younger mothers. In more recent studies, Yerushalmy[10] has demonstrated that age of father and mother influences the newborn infants' chances of survival. Stillbirth rates are lowest for children born of fathers 30 to 34 and mothers 20 to 24 years of age.

Read before the New York Society of Orthodontists, March 4, 1946.

*Papers I and IV in this series, published elsewhere, show that men with bad teeth tend to have wives whose teeth are worse than average, while men with good teeth have wives whose teeth are better than average. This is a phenomenon known as "assortative mating." Paper IV shows that the amount of caries in a child is closely related to the amount experienced by the mother and father, in spite of the fact that the parents were born in Japan and the child in the United States.

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Are parallel effects evident on the caries susceptibility of the teeth? More specifically, do children born of older parents tend to have a different susceptibility to dental caries than do children born of younger parents?

To study that question, dental examination findings on more than 1,500 children and their parents were analyzed. All the children were born in the Southern California area, of parents born in Japan. At the time the examinations were made (in 1943) the children had been relocated with their parents to the Colorado River Relocation Center at Poston, Arizona. The numbers of children aged 10 through 19 years, whose mothers and fathers were of particular ages at the time the children were born, are given in Table I. This table also gives the caries experience found in these children, expressed as the number of DMF (decayed, missing, and filled) permanent teeth per 100 erupted permanent teeth. This type of rate is used in order that the data on boys and girls may be combined on the basis of the rationale that, at the same ages, the higher caries experience of girls is the result of earlier tooth eruption in the females.[7]

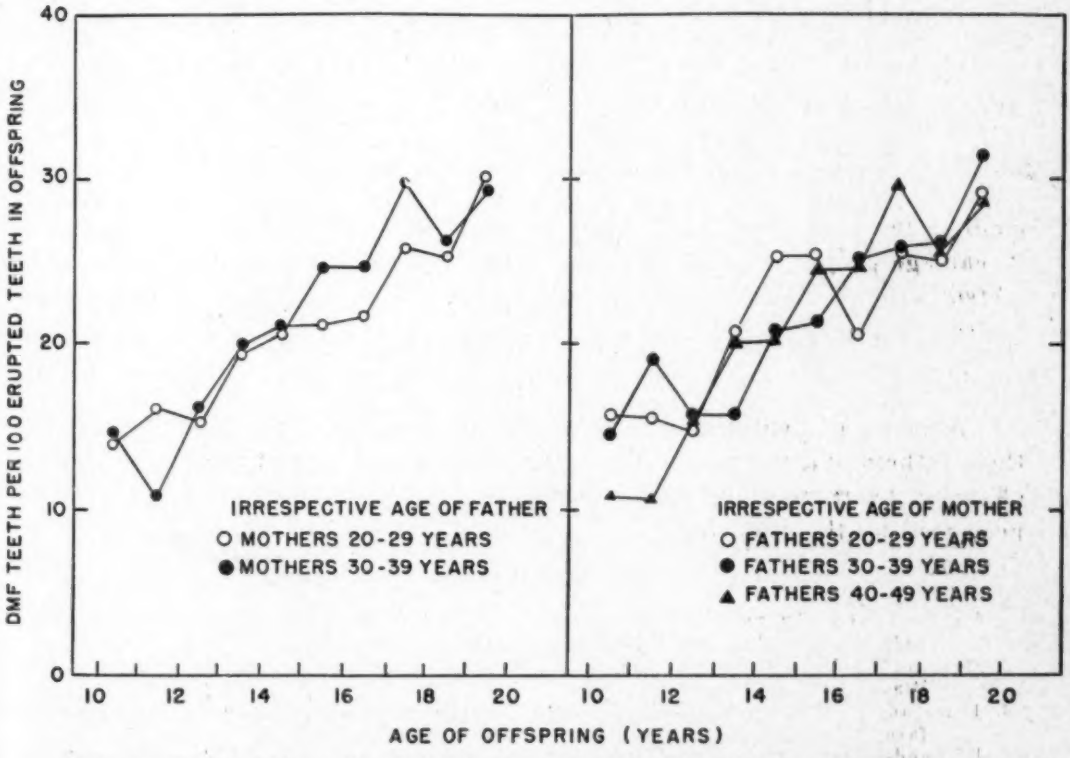

Fig. 1.—Relationship at time of birth of offspring, between number of DMF teeth per 100 erupted teeth and age, for offspring of both sexes of fathers of given ages irrespective of age of mother, and for children of both sexes of mothers of given ages irrespective of age of father.

Study of these data reveals that neither age of mother nor age of father at the time of birth of their offspring has any consistent relationship to the amount of dental decay experienced by the offspring. The differences, where they exist, are small and clearly not consistently related to age of either parent, as is shown graphically in Fig. 1. It is to be noted that the analysis is

TABLE I. NUMBER OF OFFSPRING OF SPECIFIED AGES WHOSE PARENTS WERE OF SPECIFIED AGES AT TIME OF BIRTH OF OFFSPRING, AND NUMBER OF DMF TEETH PER 100 ERUPTED TEETH AMONG SUCH OFFSPRING. (DATA BASED ON EXAMINATION OF EVACUEES OF JAPANESE ANCESTRY, RESIDING AT THE COLORADO RIVER RELOCATION CENTER, POSTON, ARIZ.)

AGE OF PARENT (YEARS) AT BIRTH OF OFFSPRING		AGE OF OFFSPRING (YEARS)									
MOTHER	FATHER	10	11	12	13	14	15	16	17	18	19
		Number of Offspring Examined									
20-29	20-29	12	12	11	22	20	25	35	27	36	18
	30-39	30	40	24	29	44	48	40	48	55	55
	40-49	5	12	13	12	24	28	36	29	27	32
30-39	30-39	11	6	12	15	30	22	13	15	11	12
	40-49	28	53	51	71	52	59	52	54	37	26
	50-59	19	17	9	12	2	12	10	9	4	5
		Number DMF Teeth per 100 Erupted Teeth in Offspring									
20-29	20-29	16.5	16.0	14.7	21.8	25.6	25.8	21.2	25.9	24.9	28.4
	30-39	13.6	19.4	15.5	17.9	22.5	22.9	23.0	23.7	26.0	31.4
	40-49	*	10.7	15.8	17.3	18.6	18.0	25.1	32.3	24.9	29.1
	All	13.6	17.1	15.4	19.1	22.1	22.2	23.1	26.7	25.4	30.2
30-39	30-39	17.4	*	17.7	13.4	20.7	22.2	31.8	35.9	37.7	42.3
	40-49	12.6	11.5	16.0	21.3	21.7	27.7	23.8	28.2	25.9	26.4
	50-59	15.4	11.7	*	19.0	*	13.7	19.3	*	*	*
	All	14.4	11.5	17.0	19.8	21.6	24.6	24.6	29.9	27.6	29.7
All	20-29	16.5	16.0	14.7	21.8	25.6	25.8	21.2	25.9	24.9	28.4
	30-39	14.6	18.5	16.3	16.4	21.8	22.6	25.2	26.6	27.9	33.4
	40-49	11.7	11.4	16.0	20.7	20.7	24.6	24.3	30.0	25.4	27.8

*Rates are not computed for age groups containing fewer than 10 children.

made in terms of 10-year age group of mothers and of fathers. Analyses using 5-year age groups, that is, 20 to 24 years, 25 to 29 years, have also been completed without finding any significant relation between age of parents and caries experience in offspring.

SUMMARY

Analysis of dental examination findings on more than 1,500 children and their fathers and mothers indicate that age of each parent at time of birth of offspring bears no significant relationship to the amount of dental decay experienced by the offspring.

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THE FAMILY AND DENTAL DISEASE

III. Size of Family and Dental Caries (DMF) Experience in Offspring

Henry Klein, D.D.S., Sc.D.,* Bethesda, Md.

IN A systematic analysis of the familial factors in dental caries susceptibility,[6-8] size of family (number of offspring) represents one variable that merits study. Socioeconomic status and fertility are inversely related; that is, the lower the economic status, the larger the family.[9] It is equally well recognized that adequacy and balance of the various items of food in the diet decline as the income becomes lower and the family larger.[1, 2] Among British children, offspring from larger families are, on the average, shorter in height and lighter in weight, with lower hemoglobin levels and weaker strength of hand grip than children from smaller families.[10] Hence, the larger families have children among whom a greater proportion have physical defects than is the case among children of smaller families.[3]

In previous publications[4, 5] it has been shown that socioeconomic status bears no significant relationship to the amount of dental caries found in white school children. The data of the present report provide additional support for that view, based on an analysis of family size and dental caries.

The findings are based on dental examination of more than 1,700 children of Japanese ancestry residing at the Colorado River Relocation Center at Poston, Arizona. Their age distributions by size of family are shown in the upper section of Table I. Their caries experience expressed as a function of the number of erupted teeth (decayed, nursing, and filled teeth per 100 erupted teeth) is shown in the lower section of Table I.

Table I. Number of Children of Specified Ages in Families Having Specified Number of Offspring, and Number of DMF Teeth per 100 Erupted Teeth Among Such Children. (Data Based on Examination of Evacuees of Japanese Ancestry, Residing at the Colorado River Relocation Center, Poston, Ariz.)

Number of offspring in family	Age (years)										
	10	11	12	13	14	15	16	17	18	19	All
	Number of Children Examined										
1, 2, or 3	27	44	32	41	26	43	49	61	54	47	424
4 or 5	39	50	39	59	74	85	87	76	71	63	643
6 or more	54	67	63	79	84	87	63	64	63	49	673
All	120	161	134	179	184	215	199	201	188	159	1740
	Number DMF Teeth per 100 Erupted Teeth										
1, 2, or 3	13.4	16.5	17.5	20.1	20.2	27.9	25.8	27.3	25.6	31.2	22.6*
4 or 5	15.0	14.2	16.1	18.9	22.8	22.0	23.4	27.0	25.9	30.4	21.6*
6 or more	13.4	12.9	17.6	21.4	22.7	21.2	23.8	28.4	26.9	27.7	21.6*
All	13.9	14.3	17.1	20.3	22.4	22.9	24.1	27.6	26.1	29.8	

*Simple arithmetic average of age-specific rates.

Read before the New York Society of Orthodontists, March 4, 1946.

*Senior Dental Officer, Division of Public Health Methods, United States Public Health Service.

As indicated in that table and in Fig. 1, size of family shows no significant or consistent relationship to the amount of caries experienced by the offspring.

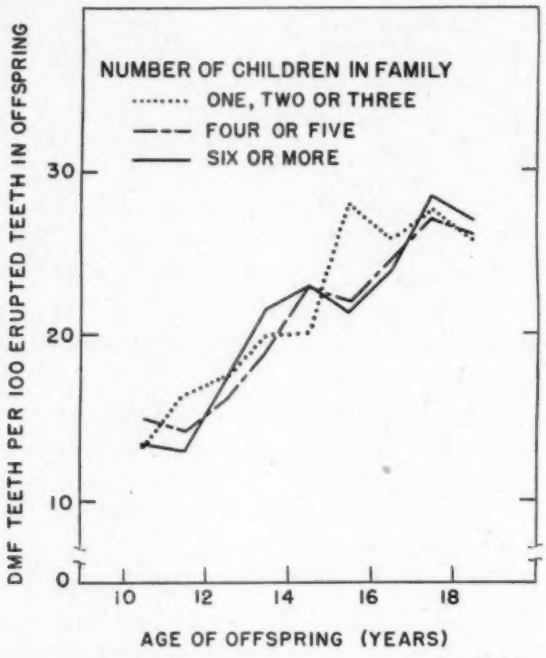

Fig. 1.—Relationship between number of DMF teeth per 100 erupted teeth and age of offspring, by size of family.

At the same age, on the average, the child of the small family (1, 2, or 3 children) shows about the same amount of dental caries experience as the child of a large family (6 or more children). On the basis of these data, analyzed in the described manner, size of family does not appear to be related significantly to dental caries susceptibility.

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Tables for Predicting Adult Height From Skeletal Age and Present Height: By Nancy Bayley, Ph.D., Berkeley, Calif., *J. Pediat.* **28**: 49-64, January, 1946.

Recent studies carried out at the University of California on the relation of growth to skeletal age indicate that it is possible to estimate, within certain limits of error, the probable adult height of children whose growth is still incomplete.

Children who mature early follow a different course of growth than children who mature late. Furthermore there are sex differences in the patterns of growth of early and late maturers. In general, for both sexes, there are two factors which influence growth. (1) During childhood, the mere differences in velocity of maturation make the rapid maturers large for their age and the slow maturers small. But (2) the longer the period of time a person is in the process of growing (that is, epiphyses open and healthy growth factors operating), the more opportunity he has for growth. Therefore, though slow maturers are small for their age, they tend, actually, to be large for their skeletal age, while the rapid maturers, though large for their chronological age, tend to be small for their skeletal age.

In girls some factor, probably related to the female sex hormones, stops growth rather abruptly after menarche. As a result we find that early maturing girls are usually large when young, slowing down to about average in height at 13 years, and completing their growth rapidly, to become small adults. Late maturing girls, conversely, are more often small when young, catch up to the average at about 13 and become tall adults.

Early maturing boys do not exhibit this abrupt curtailment of growth but slow down more gradually, and they seem to have a normal likelihood of becoming tall, or average, or short adults. Late maturing boys continue to grow, some even into their early twenties, and more often than not they become tall adults. Because of further differences in body build of early and late maturing boys, the late maturers, while young, are at a considerable disadvantage, physically: the early maturers tend to be large and broad-built or heavy-set at all ages, while the late maturers are usually slender and long-legged, their greater adult height being due primarily to continued growth of the legs.

Because of these characteristic differences between children growing at different velocities, predictions of their adult height will be more nearly accurate if determined for each group separately.

Tables designed to facilitate estimates of adult height are presented here, for boys and girls, between the ages of 7 years and maturity. These tables are constructed from data from two sources, Massachusetts children observed in the Harvard Growth Study and the California children studied at the Institute of Child Welfare of the University of California.

To secure a measure of relative growth each child's height at any given time was expressed as a percentage of his own adult stature as measured after closure of the epiphyses of the bones in his hand, wrist, and knee. When this

was done, the per cent of mature height attained was found to be very closely related to skeletal age as determined by the Todd standards. This relationship to skeletal age was found to be much closer than to chronological age. It is reasoned, therefore, that if we wish to predict adult height, the prediction will be much more accurate if a child's skeletal age is taken into account in making the estimate. This is especially true during the early teens when differences in physical maturity contribute so largely to differences in children's heights. In using these tables, it must be remembered that this system of predictions is based on measurements of normal children. We cannot expect it to be applicable to cases of extreme deviation in which the regulators of growth are not functioning normally.

The tables have been constructed to facilitate the process of computing predicted height. In order to predict a child's mature height it is necessary to have an assessment of his skeletal age (based on x-rays which have been compared with standards such as those of Todd or Flory), and his height, measured at the time the x-rays were taken. For the younger ages it is necessary, in addition, to have the child's chronological age, because age must be taken into account when a child is retarded or accelerated in a marked degree. Therefore, separate tables have been constructed for children under twelve years who deviate widely from their age norms.

DIRECTIONS FOR USING THE PREDICTION TABLES

First, select the correct table for the child's sex, skeletal age, and degree of acceleration or retardation. (Because girls mature more rapidly than boys, the difference during adolescence amounting, on the average, to two years, it is important that a child be assessed on standards and tables for his or her own sex.) The boys' tables, numbered I, are divided for convenience of handling into two age groups, Table IA for skeletal ages (Sk.A.) below 13 years, and Table IB for those 13 years and above. Two additional tables take care of the younger boys whose skeletal ages deviate widely from their chronological ages. Table IC is for boys accelerated more than one year, if their skeletal ages are 12½ years or less. Table ID is for boys retarded more than one year, if their skeletal ages are 11¾ years or less. The girls' tables numbered II are similarly divided: Table IIA for girls with skeletal ages of 11 years or less, and Table IIB for those above 11 years. The supplementary tables for the younger, more deviate girls are: Table IIC for girls who are accelerated more than one year and with skeletal ages of 14 years or less; and Table IID for girls who are retarded more than one year and whose skeletal ages are under 12 years.

Once the correct table is selected the predictions may be read directly from the table. Find the column which represents the child's skeletal age and the line which represents his height. The figure in the square where the two intersect is his predicted mature height. For example, a 10½-year-old boy who is 57 inches tall and has a bone age of 11 years should be expected, from Table IA, to have a mature height of 70.4 inches. If, however, this boy's chronological age is 8 years, 9 months, he is accelerated more than one year and, according to Table IC, his expected adult height will be 72.0 inches. On the other hand, if he is 13 years, 3 months old, with this same height and skeletal age, we might (if we did not know his skeletal age) expect him to be a very short adult (64.6 inches, Table IB), instead of the more likely 69.3 inches predicted from Table ID.

It is hoped that the tables presented here will have clinical usefulness. They should prove of value, when used with x-rays of the hand and/or other ossification centers which have been assigned skeletal ages, in predicting the adult height of children whose growth is incomplete. It seems very likely that further knowledge of the factors influencing growth will eventually make it possible to predict height with even great accuracy than the use of skeletal-age-height tables permit.

TABLE IA. FOR ESTIMATING MATURE HEIGHT OF BOYS, WITH SKELETAL AGES WITHIN ONE YEAR OF THEIR CHRONOLOGICAL AGES: SKELETAL AGES, 7 TO 13 YEARS

Skeletal Age	7-0	7-3	7-6	7-9	8-0	8-3	8-6	8-9	9-0	9-3	9-6	9-9	10-0	10-3	10-6	10-9	11-0	11-3	11-6	11-9	12-0	12-3	12-6	12-9
% of Mature Height	69.3	70.1	70.6	71.3	71.8	72.5	73.4	74.3	75.0	75.8	76.5	77.4	78.1	78.9	79.7	80.3	81.0	81.4	82.7	83.0	83.8	84.4	85.5	86.4
Ht. (inches)																								
42																								
43	62.0	61.3																						
44	63.5	62.8	62.3	61.7	61.3																			
45	64.9	64.2	63.7	63.1	62.7																			
46	66.4	65.6	65.2	64.5	64.1	63.4	62.7	61.9	61.3															
47	67.8	67.0	66.6	65.9	65.4	64.8	64.0	63.3	62.7	62.0	61.4	60.7												
48	69.3	68.5	68.0	67.3	66.9	66.2	65.4	64.6	64.0	63.3	62.7	62.0												
49	70.7	69.9	69.4	68.7	68.2	67.6	66.8	65.9	65.3	64.6	64.0	63.3	62.7	62.1	61.5	61.0	60.5							
50	72.2	71.3	70.8	70.1	69.6	69.0	68.1	67.3	66.7	66.0	65.4	64.6	64.1	63.4	62.7	62.3	61.7							
51	73.6	72.8	72.2	71.5	71.0	70.3	69.5	68.6	68.0	67.3	66.7	65.9	65.4	64.6	64.0	63.5	63.0	62.7	61.7	61.4	60.9			
52	75.0	74.2	73.7	72.9	72.4	71.7	70.8	70.0	69.3	68.6	68.0	67.2	66.7	65.9	65.2	64.8	64.2	63.9	62.9	62.6	62.1	61.6		
53	76.5	75.6	75.1	74.3	73.8	73.1	72.2	71.3	70.7	69.9	69.3	68.5	67.9	67.2	66.5	66.0	65.4	65.1	64.1	63.9	63.2	62.8		
54				75.7	75.2	74.5	73.6	72.7	72.0	71.2	70.6	69.8	69.2	68.4	67.8	67.2	66.7	66.3	65.3	65.1	64.4	64.0	63.2	62.5
55				77.1	76.6	75.9	74.9	74.0	73.3	72.6	71.9	71.1	70.5	69.7	69.0	68.5	67.9	67.6	66.5	66.3	65.6	65.2	64.3	63.7
56				78.5	78.0	77.2	76.3	75.4	74.7	73.9	73.2	72.4	71.8	71.0	70.3	69.7	69.1	68.8	67.7	67.5	66.8	66.4	65.5	64.8
57								76.7	76.0	75.2	74.5	73.6	73.1	72.2	71.5	71.0	70.4	70.0	68.9	68.7	68.0	67.5	66.7	66.0
58								78.1	77.3	76.5	75.8	74.9	74.4	73.5	72.7	72.2	71.6	71.3	70.1	69.9	69.2	68.7	67.8	67.1
59												76.2	75.6	74.8	74.0	73.5	72.8	72.5	71.3	71.1	70.4	69.9	69.0	68.3
60												77.5	76.8	76.1	75.3	74.7	74.1	73.7	72.6	72.3	71.6	71.1	70.2	69.4
61															76.5	76.0	75.3	74.9	73.8	73.5	72.8	72.3	71.3	70.6
62																	76.5	76.2	75.0	74.7	74.0	73.5	72.5	71.8
63																			76.2	75.9	75.2	74.6	73.7	72.9
64																				77.1	76.4	75.8	74.9	74.1
65																				78.3	77.6	77.0	76.0	75.2
66																				79.5	78.8	78.2	77.2	76.4

TABLE IB. FOR ESTIMATING MATURE HEIGHT OF BOYS, WITH SKELETAL AGES, 13 YEARS TO MATURITY

Skeletal Age	13-0	13-3	13-6	13-9	14-0	14-3	14-6	14-9	15-0	15-3	15-6	15-9	16-0	16-3	16-6	16-9	17-0	17-3	17-6	17-9	18-0	18-3	18-6	18-9
% of Mature Height	87.3	88.3	89.3	90.4	91.5	92.7	93.7	94.5	95.5	96.3	96.9	97.3	97.7	98.2	98.3	98.5	98.8	99.0	99.2	99.4	99.6	99.8	99.9	100
Ht. (inches)																								
54	61.9																							
55	63.0	62.3	61.6																					
56	64.1	63.4	62.7	61.9																				
57	65.3	64.6	63.8	63.0	62.3	61.5	60.8	60.3																
58	66.4	65.7	64.9	64.2	63.4	62.6	61.9	61.4																
59	67.6	66.8	66.1	65.3	64.5	63.6	63.0	62.4																
60	68.7	68.0	67.2	66.4	65.6	64.7	64.0	63.5	62.8	62.3	61.9	61.7	61.4											
61	69.9	69.1	68.3	67.5	66.7	65.8	65.1	64.6	63.9	63.3	63.0	62.7	62.4											
62	71.0	70.2	69.4	68.6	67.8	66.9	66.2	65.6	64.9	64.4	64.0	63.7	63.4											
63	72.2	71.3	70.5	69.7	68.9	68.0	67.2	66.7	66.0	65.4	65.0	64.7	64.5											
64	73.3	72.5	71.7	70.8	69.9	69.0	68.3	67.7	67.0	66.5	66.0	65.8	65.5	65.2	65.1	65.0	64.8	64.6	64.5	64.4	64.3	64.1	64.1	64.0
65	74.5	73.6	72.8	71.9	71.0	70.1	69.4	68.8	68.1	67.5	67.1	66.8	66.5	66.2	66.1	66.0	65.8	65.7	65.5	65.4	65.3	65.1	65.1	65.0
66	75.6	74.7	73.9	73.0	72.1	71.2	70.4	69.8	69.1	68.5	68.1	67.8	67.6	67.2	67.1	67.0	66.8	66.7	66.5	66.4	66.3	66.1	66.1	66.0
67	76.7	75.9	75.0	74.1	73.2	72.3	71.5	70.9	70.2	69.6	69.1	68.9	68.6	68.2	68.2	68.0	67.8	67.7	67.5	67.4	67.3	67.1	67.1	67.0
68	77.9	77.0	76.1	75.2	74.3	73.4	72.6	72.0	71.2	70.6	70.2	69.9	69.6	69.2	69.2	69.0	68.8	68.7	68.5	68.4	68.3	68.1	68.1	68.0
69	79.0	78.1	77.3	76.3	75.4	74.4	73.6	73.0	72.3	71.7	71.2	70.9	70.6	70.3	70.2	70.0	69.8	69.7	69.6	69.4	69.3	69.1	69.1	69.0
70	80.2	79.3	78.4	77.4	76.5	75.5	74.7	74.1	73.3	72.7	72.2	71.9	71.6	71.3	71.2	71.1	70.8	70.7	70.6	70.4	70.3	70.1	70.1	70.0
71					77.6	76.6	75.8	75.1	74.3	73.7	73.3	73.0	72.7	72.3	72.2	72.1	71.9	71.7	71.6	71.4	71.3	71.1	71.1	71.0
72						77.7	76.8	76.2	75.4	74.8	74.3	74.0	73.7	73.3	73.3	73.1	72.9	72.7	72.6	72.4	72.3	72.1	72.1	72.0
73						78.7	77.9	77.2	76.4	75.8	75.3	75.0	74.7	74.3	74.3	74.1	73.9	73.7	73.6	73.4	73.3	73.1	73.1	73.0
74							79.0	78.3	77.5	76.8	76.4	76.0	75.7	75.4	75.3	75.1	74.9	74.7	74.6	74.4	74.3	74.1	74.1	74.0
75															76.3	76.1	75.9	75.7	75.6	75.4	75.3	75.2	75.1	75.0
76																	76.9	76.7	76.6	76.4	76.3	76.2	76.1	76.0
77																	77.9	77.7	77.6	77.4	77.3	77.2	77.1	77.0
78																	78.9	78.7	78.6	78.4	78.3	78.2	78.1	78.0

TABLE IC. FOR ESTIMATING MATURE HEIGHT OF BOYS ACCELERATED MORE THAN ONE YEAR, BUT WITH SKELETAL AGES UNDER 13 YEARS

Skeletal Age	7-0	7-3	7-6	7-9	8-0	8-3	8-6	8-9	9-0	9-3	9-6	9-9	10-0	10-3	10-6	10-9	11-0	11-3	11-6	11-9	12-0	12-3	12-6
% of Mature Height	67.0	67.6	68.2	68.9	69.4	70.1	71.0	71.9	72.6	73.4	74.1	75.1	75.9	76.8	77.7	78.3	79.2	80.0	80.8	81.5	82.5	83.5	84.5
Ht. (inches)																							
43	64.2	63.6																					
44	65.7	65.1	64.5	63.9																			
45	67.2	66.6	66.0	65.3																			
46	68.7	68.0	67.4	66.8																			
47	70.1	69.5	68.9	68.2	67.7	67.0	66.2	65.4															
48	71.6	71.0	70.4	69.7	69.2	68.5	67.6	66.8	66.1	65.4	64.8												
49	73.1	72.5	71.8	71.1	70.6	69.9	69.0	68.2	67.5	66.8	66.1	65.2	64.6										
50	74.6	74.0	73.3	72.6	72.0	71.3	70.4	69.5	68.9	68.1	67.5	66.6	65.9	65.1	64.4	63.9							
51	76.1	75.4	74.8	74.0	73.5	72.8	71.8	70.9	70.2	69.5	68.8	67.9	67.2	66.4	65.6	65.1							
52	77.6	76.9	76.2	75.5	74.9	74.2	73.2	72.3	71.6	70.8	70.2	69.2	68.5	67.7	66.9	66.4	65.7	65.0	64.4	63.8			
53		78.4	77.7	76.9	76.4	75.6	74.6	73.7	73.0	72.2	71.5	70.6	69.8	69.0	68.2	67.7	66.9	66.2	65.0	65.0	64.2	63.5	62.7
54					77.8	77.0	76.1	75.1	74.4	73.6	72.9	71.9	71.1	70.3	69.5	69.0	68.2	67.5	66.8	66.3	65.5	64.7	63.9
55							77.5	76.5	75.8	74.9	74.2	73.2	72.5	71.6	70.9	70.2	69.4	68.8	68.1	67.5	66.7	65.9	65.1
56									77.1	76.3	75.6	74.6	73.8	72.9	72.1	71.5	70.7	70.0	69.3	68.7	67.9	67.1	66.3
57									78.5	77.7	76.9	75.9	75.1	74.2	73.4	72.8	72.0	71.2	70.5	69.9	69.1	68.3	67.5
58											78.3	77.2	76.4	75.5	74.6	74.1	73.2	72.5	71.8	71.2	70.3	69.5	68.6
59													77.7	76.8	75.9	75.3	74.5	73.8	73.0	72.4	71.5	70.7	69.8
60														78.1	77.2	76.6	75.8	75.0	74.3	73.6	72.7	71.9	71.0
61																	77.0	76.2	75.5	74.8	73.9	73.1	72.2
62																	78.3	77.5	76.7	76.1	75.2	74.3	73.4
63																			78.0	77.3	76.4	75.4	74.6
64																				78.5	77.6	76.6	75.7
65																							76.9

NOTE: If deviation in skeletal age is more than two years, estimate is height predicted from this table, "or taller."

TABLE ID. FOR ESTIMATING MATURE HEIGHT OF BOYS RETARDED MORE THAN ONE YEAR BUT WITH SKELETAL AGES UNDER 12

Skeletal Age	6-0	6-3	6-6	6-9	7-0	7-3	7-6	7-9	8-0	8-3	8-6	8-9	9-0	9-3	9-6	9-9	10-0	10-3	10-6	10-9	11-0	11-3	11-6	11-9
% of Mature Height	69.5	69.8	70.3	71.0	71.6	72.5	73.0	73.7	74.2	74.9	75.8	76.7	77.4	78.2	78.9	79.7	80.3	81.0	81.2	81.8	82.2	83.0	83.5	84.0
Ht. (inches)																								
43	61.9	61.6	61.2	60.6	60.0																			
44	63.3	63.0	62.6	62.0	61.4	60.7	60.3																	
45	64.7	64.5	64.0	63.4	62.8	62.1	61.6	61.0																
46	66.2	65.9	65.4	64.8	64.2	63.4	63.0	62.4	62.0	61.4	60.7	60.0												
47	67.6	67.3	66.9	66.2	65.6	64.8	64.4	63.8	63.3	62.8	62.0	61.3												
48	69.1	68.8	68.3	67.6	67.0	66.2	65.8	65.1	64.7	64.1	63.3	62.6	62.0	61.4	60.8	60.2								
49	70.5	70.2	69.7	69.0	68.4	67.6	67.1	66.5	66.0	65.4	64.6	63.9	63.3	62.7	62.1	61.5								
50	71.9	71.6	71.1	70.4	69.8	69.0	68.5	67.8	67.4	66.8	66.0	65.2	64.6	63.9	63.4	62.7	62.3	61.7	61.6	61.1				
51	73.4	73.1	72.5	71.8	71.2	70.3	69.9	69.2	68.7	68.1	67.3	66.5	65.9	65.2	64.6	64.0	63.5	63.0	62.8	62.3				
52					72.6	71.7	71.2	70.6	70.1	69.4	68.6	67.8	67.2	66.5	65.9	65.2	64.8	64.2	64.0	63.6	63.3	62.7	62.3	61.9
53					74.0	73.1	72.6	71.9	71.4	70.8	69.9	69.1	68.5	67.8	67.2	66.5	66.0	65.4	65.3	64.8	64.5	63.9	63.5	63.1
54								73.3	72.8	72.1	71.2	70.4	69.8	69.1	68.4	67.8	67.2	66.7	66.5	66.0	65.7	65.0	64.7	64.3
55									74.1	73.4	72.6	71.7	71.1	70.3	69.7	69.0	68.5	67.9	67.7	67.2	66.9	66.3	65.9	65.5
56											73.9	73.0	72.4	71.6	71.0	70.3	69.7	69.1	69.0	68.5	68.1	67.5	67.1	66.7
57													73.6	72.9	72.2	71.5	71.0	70.4	70.2	69.7	69.3	68.7	68.3	67.9
58															73.5	72.8	72.2	71.6	71.4	70.9	70.6	69.9	69.5	69.0
59																	73.5	72.8	72.7	72.1	71.8	71.1	70.7	70.2
60																		74.1	73.9	73.3	73.0	72.3	71.9	71.4
61																				74.6	74.2	73.5	73.1	72.6
62																					75.4	74.7	74.3	73.8
63																								75.0

NOTE: If deviation in skeletal age is more than two years, estimate is height predicted from this table, "or shorter."

TABLE IIA. FOR ESTIMATING MATURE HEIGHT OF GIRLS WITH SKELETAL AGES WITHIN ONE YEAR OF THEIR CHRONOLOGICAL AGES: SKELETAL AGES FROM 6 THROUGH 11 YEARS

Skeletal Age	6-0	6-3	6-6	6-9	7-0	7-3	7-6	7-9	8-0	8-3	8-6	8-9	9-0	9-3	9-6	9-9	10-0	10-3	10-6	10-9	11-0
% of Mature Height	72.0	72.2	72.7	73.4	74.1	75.0	76.1	76.7	77.5	78.2	79.0	79.8	81.0	81.3	82.2	83.1	84.0	84.9	85.6	86.2	87.2
Ht. (inches)																					
40	55.6	55.4	55.0	54.5																	
41	56.9	56.8	56.4	55.9																	
42	58.3	58.2	57.8	57.2																	
43	59.7	59.6	59.1	58.6	58.0	57.3	56.5														
44	61.1	60.9	60.5	59.9	59.4	58.7	57.8	57.4	56.8	56.3	55.7	55.1	54.3	54.1	53.5	52.9	52.4	51.8			
45	62.5	62.3	61.9	61.3	60.7	60.0	59.1	58.7	58.1	57.5	57.0	56.4	55.6	55.4	54.7	54.2	53.6	53.0	52.6	52.2	
46	63.9	63.7	63.3	62.7	62.1	61.3	60.4	60.0	59.4	58.8	58.2	57.6	56.8	56.6	56.0	55.4	54.8	54.2	53.7	53.4	52.8
47	65.3	65.1	64.6	64.0	63.4	62.7	61.8	61.3	60.6	60.1	59.5	58.9	58.0	57.8	57.2	56.6	56.0	55.4	54.9	54.5	53.9
48	66.7	66.5	66.0	65.4	64.8	64.0	63.1	62.6	61.9	61.4	60.8	60.2	59.3	59.0	58.4	57.8	57.1	56.5	56.1	55.7	55.0
49	68.1	67.9	67.4	66.8	66.1	65.3	64.4	63.9	63.2	62.7	62.0	61.4	60.5	60.3	59.6	59.0	58.3	57.7	57.2	56.8	56.2
50	69.4	69.3	68.8	68.1	67.5	66.7	65.7	65.2	64.5	63.9	63.3	62.7	61.7	61.5	60.8	60.2	59.5	58.9	58.4	58.0	57.3
51				69.5	68.8	68.0	67.0	66.5	65.8	65.2	64.6	63.9	63.0	62.7	62.0	61.4	60.7	60.1	59.6	59.2	58.5
52				70.8	70.2	69.3	68.3	67.8	67.1	66.5	65.8	65.2	64.2	64.0	63.3	62.6	61.9	61.2	60.7	60.3	59.6
53					71.5	70.7	69.6	69.1	68.4	67.8	67.1	66.4	65.4	65.2	64.5	63.8	63.1	62.4	61.9	61.5	60.8
54							71.0	70.4	69.7	69.1	68.4	67.7	66.7	66.4	65.7	65.0	64.3	63.6	63.1	62.6	61.9
55									71.0	70.3	69.6	68.9	67.9	67.7	66.9	66.2	65.5	64.8	64.3	63.8	63.1
56									72.3	71.6	70.9	70.2	69.1	68.9	68.1	67.4	66.7	66.0	65.4	65.0	64.2
57											72.2	71.4	70.4	70.1	69.3	68.6	67.9	67.1	66.6	66.1	65.4
58													71.6	71.3	70.6	69.8	69.0	68.3	67.8	67.3	66.5
59														72.6	71.8	71.0	70.2	69.5	68.9	68.4	67.7
60																72.2	71.4	70.7	70.1	69.6	68.8
61																	72.6	71.8	71.3	70.8	70.0
62																			72.4	71.9	71.1
63																				73.1	72.2

TABLE IIB. FOR ESTIMATING MATURE HEIGHT OF GIRLS WITH SKELETAL AGES BETWEEN 11 YEARS AND MATURITY

Skeletal Age	11-3	11-6	11-9	12-0	12-3	12-6	12-9	13-0	13.3	13-6	13-9	14-0	14-3	14-6	14-9	15-0	15-3	15-6	15-9	16-0	16-3	16-6
% of Mature Height	88.0	89.2	90.3	91.7	92.7	93.6	94.7	95.5	96.5	97.2	97.7	98.0	98.5	98.6	98.9	99.0	99.1	99.4	99.6	99.8	99.9	100
Ht. (inches)																						
46	52.3																					
47	53.4	52.7	52.0																			
48	54.5	53.8	53.2	52.3	51.8																	
49	55.7	54.9	54.3	53.4	52.9	52.4	51.7															
50	56.8	56.0	55.4	54.5	53.9	53.4	52.8															
51	58.0	57.2	56.5	55.6	55.0	54.5	53.9	53.4	52.8	52.5	52.2											
52	59.1	58.3	57.6	56.7	56.1	55.6	54.9	54.4	53.9	53.5	53.2											
53	60.2	59.4	58.7	57.8	57.2	56.6	56.0	55.5	54.9	54.5	54.2	54.1	53.8	53.8								
54	61.4	60.5	59.8	58.9	58.3	57.7	57.0	56.5	56.0	55.6	55.3	55.1	54.8	54.8								
55	62.5	61.7	60.9	60.0	59.3	58.8	58.1	57.6	57.0	56.6	56.3	56.1	55.8	55.8	55.6	55.6	55.5	55.3				
56	63.6	62.8	62.0	61.1	60.4	59.8	59.1	58.6	58.0	57.6	57.3	57.1	56.9	56.8	56.6	56.6	56.5	56.3	56.2	56.1	56.1	56.0
57	64.8	63.9	63.1	62.2	61.5	60.9	60.2	59.7	59.1	58.6	58.3	58.2	57.9	57.8	57.6	57.6	57.5	57.3	57.2	57.1	57.1	57.0
58	65.9	65.0	64.2	63.2	62.6	62.0	61.2	60.7	60.1	59.7	59.4	59.2	58.9	58.8	58.6	58.6	58.5	58.4	58.2	58.1	58.1	58.0
59	67.0	66.1	65.3	64.3	63.6	63.0	62.3	61.8	61.1	60.7	60.4	60.2	59.9	59.8	59.7	59.6	59.5	59.4	59.2	59.1	59.1	59.0
60	68.2	67.3	66.4	65.4	64.7	64.1	63.4	62.8	62.2	61.7	61.4	61.2	60.9	60.9	60.7	60.6	60.5	60.4	60.2	60.1	60.1	60.0
61	69.3	68.4	67.6	66.5	65.8	65.2	64.4	63.9	63.2	62.8	62.4	62.2	61.9	61.9	61.7	61.6	61.6	61.4	61.2	61.1	61.1	61.0
62	70.5	69.5	68.7	67.6	66.9	66.2	65.5	64.9	64.3	63.8	63.5	63.3	62.9	62.9	62.7	62.6	62.6	62.4	62.2	62.1	62.1	62.0
63	71.6	70.6	69.8	68.7	68.0	67.3	66.5	66.0	65.3	64.8	64.5	64.3	64.0	63.9	63.7	63.6	63.6	63.4	63.3	63.1	63.1	63.0
64		71.7	70.9	69.8	69.0	68.4	67.6	67.0	66.3	65.8	65.5	65.3	65.0	64.9	64.7	64.6	64.6	64.4	64.3	64.1	64.1	64.0
65				70.9	70.1	69.4	68.6	68.1	67.4	66.9	66.5	66.3	66.0	65.9	65.7	65.7	65.6	65.4	65.3	65.1	65.1	65.0
66						70.5	69.7	69.1	68.4	67.9	67.6	67.3	67.0	66.9	66.7	66.7	66.6	66.4	66.3	66.1	66.1	66.0
67						71.6	70.7	70.2	69.4	68.9	68.6	68.4	68.0	68.0	67.7	67.7	67.6	67.4	67.3	67.1	67.1	67.0
68								71.2	70.5	70.0	69.6	69.4	69.0	69.0	68.8	68.7	68.6	68.4	68.3	68.1	68.1	68.0
69								72.3	71.5	71.0	70.6	70.4	70.1	70.0	69.8	69.7	69.6	69.4	69.3	69.1	69.1	69.0
70											71.6	71.4	71.1	71.0	70.8	70.7	70.6	70.4	70.3	70.1	70.1	70.0
71											72.7	72.4	72.1	72.0	71.8	71.7	71.6	71.4	71.3	71.1	71.1	71.0
72											73.7	73.5	73.1	73.0	72.8	72.7	72.7	72.4	72.3	72.1	72.1	72.0

TABLE IIC. FOR ESTIMATING MATURE HEIGHT OF GIRLS ACCELERATED MORE THAN ONE YEAR, BUT WITH SKELETAL AGES OF 14 YEARS OR LESS

Skeletal Age	7-0	7-3	7-6	7-9	8-0	8-3	8-6	8-9	9-0	9-3	9-6	9-9	10-0	10-3	10-6	10-9	11-0	11-3	11-6	11-9	12-0	12-3	12-6	12-9	13-0	13-3	13-6	13-9	14-0
% of Mature Height	71.6	72.8	73.7	74.6	75.4	76.1	77.0	77.9	78.6	79.3	80.3	81.0	82.0	82.8	83.6	84.6	85.3	86.5	87.3	88.5	89.8	90.8	91.8	93.1	94.1	95.5	96.3	97.0	97.5
Ht. (inches)																													
43	60.1	59.1	58.3																										
44	61.5	60.4	59.7	59.0	58.4	57.8	57.1	56.5	56.0	55.5	54.8	54.3	53.7	53.1															
45	62.8	61.8	61.1	60.3	59.7	59.1	58.4	57.8	57.3	56.7	56.0	55.6	54.9	54.3	53.8	53.2													
46	64.2	63.2	62.4	61.7	61.0	60.4	59.7	59.0	58.5	58.0	57.3	56.8	56.1	55.6	55.0	54.4	53.9	53.2											
47	65.6	64.6	63.8	63.0	62.3	61.8	61.0	60.3	59.8	59.3	58.5	58.0	57.3	56.8	56.2	55.6	55.1	54.3	53.8	53.1									
48	67.0	65.9	65.1	64.3	63.7	63.1	62.3	61.6	61.1	60.5	59.8	59.3	58.5	58.0	57.4	56.7	56.3	55.5	55.0	54.2	53.5	52.9	52.3						
49	68.4	67.3	66.5	65.7	65.0	64.4	63.6	62.9	62.3	61.8	61.0	60.5	59.8	59.2	58.6	57.9	57.4	56.6	56.1	55.4	54.6	54.0	53.4	52.6					
50	69.8	68.7	67.8	67.0	66.3	65.7	64.9	64.2	63.6	63.1	62.3	61.7	61.0	60.4	59.8	59.1	58.6	57.8	57.3	56.5	55.7	55.1	54.5	53.7					
51	71.2	70.1	69.2	68.4	67.6	67.0	66.2	65.5	64.9	64.3	63.5	63.0	62.2	61.6	61.0	60.3	59.8	59.0	58.4	57.6	56.8	56.2	55.6	54.8	54.2	53.4	53.0	52.6	52.3
52	72.6	71.4	70.6	69.7	69.0	68.3	67.5	66.8	66.2	65.6	64.8	64.2	63.4	62.8	62.2	61.5	61.0	60.1	59.6	58.8	57.9	57.3	56.6	55.9	55.3	54.4	54.0	53.6	53.3
53			71.9	71.0	70.3	69.6	68.8	68.0	67.4	66.8	66.0	65.4	64.6	64.0	63.4	62.6	62.1	61.3	60.7	59.9	59.0	58.4	57.7	56.9	56.3	55.5	55.0	54.6	54.4
54				72.4	71.6	71.0	70.1	69.3	68.7	68.1	67.2	66.7	65.9	65.2	64.6	63.8	63.3	62.4	61.9	61.0	60.1	59.5	58.8	58.0	57.4	56.5	56.1	55.7	55.4
55						72.3	71.4	70.6	70.0	69.4	68.5	67.9	67.1	66.4	65.8	65.0	64.5	63.6	63.0	62.1	61.2	60.6	59.9	59.1	58.4	57.6	57.1	56.7	56.4
56								71.9	71.2	70.6	69.7	69.1	68.3	67.6	67.0	66.2	65.7	64.7	64.1	63.3	62.4	61.7	61.0	60.2	59.5	58.6	58.2	57.7	57.4
57										71.9	71.0	70.4	69.5	68.8	68.2	67.4	66.8	65.9	65.3	64.4	63.5	62.8	62.1	61.2	60.6	59.7	59.2	58.8	58.5
58											72.2	71.6	70.7	70.0	69.4	68.6	68.0	67.1	66.4	65.5	64.6	63.9	63.2	62.3	61.6	60.7	60.2	59.8	59.5
59													72.0	71.3	70.6	69.7	69.2	68.2	67.6	66.7	65.7	65.0	64.3	63.4	62.7	61.8	61.3	60.8	60.5
60															71.8	70.9	70.3	69.4	68.7	67.8	66.8	66.1	65.4	64.4	63.8	62.8	62.3	61.9	61.5
61																72.1	71.5	70.5	69.9	68.9	67.9	67.2	66.4	65.5	64.8	63.9	63.3	62.9	62.6
62																	72.7	71.7	71.0	70.1	69.0	68.3	67.5	66.6	65.9	64.9	64.4	63.9	63.6
63																		72.8	72.2	71.2	70.2	69.4	68.6	67.7	67.0	66.0	65.4	64.9	64.6
64																				72.3	71.3	70.5	69.7	68.7	68.0	67.0	66.5	66.0	65.6
65																						71.6	70.8	69.8	69.1	68.1	67.5	67.0	66.7
66																							71.9	70.9	70.1	69.1	68.5	68.0	67.7
67																								72.0	71.2	70.2	69.6	69.1	68.7
68																									72.3	71.2	70.6	70.1	69.7

TABLE IID. FOR ESTIMATING MATURE HEIGHT OF GIRLS RETARDED MORE THAN ONE YEAR, BUT WITH SKELETAL AGES UNDER 12 YEARS

Skeletal Age	6-0	6-3	6-6	6-9	7-0	7-3	7-6	7-9	8-0	8-3	8-6	8-9	9-0	9-3	9-6	9-9	10-0	10-3	10-6	10-9	11-0	11-3	11-6	11-9
% of Mature Height	74.2	74.5	75.0	75.7	76.5	77.4	78.0	78.7	79.8	80.5	81.3	82.0	82.8	83.8	84.5	85.2	86.1	86.8	87.6	88.0	88.2	89.0	90.0	91.0
Ht. (inches)																								
40	53.9	53.7	53.3	52.8																				
41	55.3	55.0	54.7	54.2																				
42	56.6	56.4	56.0	55.5																				
43	58.0	57.7	57.3	56.8	56.2	55.6	55.1																	
44	59.3	59.1	58.7	58.1	57.5	56.8	56.4	55.9	55.1	54.7	54.1	53.7	53.1	52.5	52.1	51.6	51.1	50.7						
45	60.6	60.4	60.0	59.4	58.8	58.1	57.7	57.2	56.4	55.9	55.4	54.9	54.3	53.7	53.2	52.8	52.3	51.8	51.4	51.1				
46	62.0	61.7	61.3	60.7	60.1	59.4	59.0	58.4	57.6	57.1	56.6	56.1	55.6	54.9	54.4	54.0	53.4	53.0	52.5	52.3	52.2	51.7		
47	63.3	63.1	62.7	62.1	61.4	60.7	60.3	59.7	58.9	58.4	57.8	57.3	56.8	56.1	55.6	55.2	54.6	54.1	53.7	53.4	53.3	52.8	52.2	51.6
48	64.5	64.4	64.0	63.4	62.7	62.0	61.5	61.0	60.2	59.6	59.0	58.5	58.0	57.3	56.8	56.3	55.7	55.3	54.8	54.5	54.4	53.9	53.3	52.7
49	66.0	65.8	65.3	64.7	64.1	63.3	62.8	62.3	61.4	60.9	60.3	59.8	59.2	58.5	58.0	57.5	56.9	56.5	55.9	55.7	55.6	55.1	54.4	53.8
50	67.4	67.1	66.7	66.1	65.4	64.6	64.1	63.5	62.7	62.1	61.5	61.0	60.4	59.7	59.2	58.7	58.1	57.6	57.1	56.8	56.7	56.2	55.6	54.9
51				67.4	66.7	65.9	65.4	64.8	63.9	63.4	62.7	62.2	61.6	60.9	60.4	59.9	59.2	58.8	58.2	58.0	57.8	57.3	56.7	56.0
52				68.7	68.0	67.2	66.7	66.1	65.2	64.6	64.0	63.4	62.8	62.1	61.5	61.0	60.4	59.9	59.4	59.1	59.0	58.4	57.8	57.1
53					69.3	68.5	67.9	67.3	66.4	65.8	65.2	64.6	64.0	63.2	62.7	62.2	61.6	61.1	60.5	60.2	60.1	59.6	58.9	58.2
54							69.2	68.6	67.7	67.1	66.4	65.9	65.2	64.4	63.9	63.4	62.7	62.2	61.6	61.4	61.2	60.7	60.0	59.3
55									68.9	68.3	67.7	67.1	66.4	65.6	65.1	64.6	63.9	63.4	62.8	62.5	62.4	61.8	61.1	60.4
56										69.6	68.9	68.3	67.6	66.8	66.3	65.7	65.0	64.5	63.9	63.6	63.5	62.9	62.2	61.5
57											70.1	69.5	68.8	68.0	67.5	66.9	66.2	65.7	65.1	64.8	64.6	64.0	63.3	62.6
58													70.0	69.2	68.6	68.1	67.4	66.8	66.2	65.9	65.8	65.2	64.4	63.7
59														70.4	69.8	69.2	68.5	68.0	67.4	67.0	66.9	66.3	65.6	64.8
60																	69.6	69.1	68.5	68.2	68.0	67.4	66.7	65.9
61																	70.8	70.3	69.6	69.3	69.2	68.5	67.8	67.0
62																			70.8	70.5	70.3	69.7	68.9	68.1
63																						70.8	70.0	69.2
64																							71.1	70.3

NOTE: If deviation in skeletal age is more than two years, estimate is height predicted from this table, "or shorter."

News and Notes

TENTATIVE PROGRAM FOR THE MEETING OF THE AMERICAN ASSOCIATION OF ORTHODONTISTS IN COLORADO SPRINGS, COLORADO

SEPT. 30, OCT. 1, 2, 3, 1946

Monday, Sept. 30, 1946

ROCKY MOUNTAIN SOCIETY OF ORTHODONTISTS DAY

Henry F. Hoffman, President of the Rocky Mountain Society, presiding

MORNING:

9:00 Invocation.
Address of Welcome. Lester C. Hunt, D.D.S., Governor of the State of Wyoming.
Response to Address of Welcome. Earl G. Jones, President-Elect, Columbus, Ohio.

9:15 President's Address. Archie B. Brusse, Denver, Colorado.

9:45 The Evolution and Devolution of the Human Face. Ernest A. Hooton, Professor of Anthropology, Harvard University.

11:00 "Use of the Twin Arch Mechanism in the Treatment of Cases in Which Extraction Is Indicated." Joseph Johnson, Louisville, Kentucky.

12:00 Luncheon.

AFTERNOON:

Lecture Clinics (clinics will be repeated three times during the afternoon).

"Removable Appliance." S. D. Gore, New Orleans, Louisiana.

"Tooth Positioner." Harold D. Kesling, La Porte, Indiana.

"Longtube X-ray Technic." Gordon Fitzgerald.

Tuesday, Oct. 1, 1946

PACIFIC COAST SOCIETY OF ORTHODONTISTS DAY

J. Camp Dean, President of the Pacific Coast Society, presiding.

MORNING:

9:00 "The Limitations of Orthodontic Treatment." Part 2, "Permanent Dentition Diagnosis and Treatment." Hays N. Nance, Pasadena, California.
(Part 1 was read before the Southern Society of Orthodontists on Jan. 28, 1946.)
"Gnathostatic Diagnosis." Will G. Sheffer, San Jose, California.
"The Principles and Mechanics of Treatment With the Sliding Twin Section Appliance." Clarence Carey, Palo Alto, California.

12:00 Round Table luncheon presided over by George W. Hahn, Berkeley, California (who will be remembered for the way in which he conducted the symposium at the Chicago meeting).

Following luncheon there will be an afterluncheon speech by Spencer Atkinson, Pasadena, California, entitled "History, Trends, and Future of Orthodontics."

Wednesday, Oct. 2, 1946

SOUTHWESTERN SOCIETY OF ORTHODONTISTS DAY

Brooks Bell, President of the Southwestern Society, presiding.

MORNING:

9:00 "Development of the Mandible." Henry Sicher, Chicago, Illinois.
11:00 "Anthropometry and Orthodontia." Ernest A. Hooton, Harvard University.
12:00 Luncheon

AFTERNOON

2:00 Lecture Clinics (clinics will be repeated twice during the afternoon).
"Labial Lingual Arch Technic and the Guide Plane."
Oren Oliver, Nashville, Tennessee.
Russell Irish, Pittsburgh, Pennsylvania.
"Edgewise Mechanism."
Edward Arnold, Houston, Texas.
A. P. Westfall, Houston, Texas.
Bert Gaylord, Dallas, Texas.
C. G. Rowland, San Antonio, Texas.

Thursday, Oct. 3, 1946

MORNING:

9:30 Research Section, under the direction of Allan G. Brodie, Chicago, Illinois.
10:00 Prize Essay.
11:00 Business Meeting.

Change in Meeting Place, American Board of Orthodontics

The headquarters of the American Board of Orthodontics has been changed from the Broadmoor Hotel to the Antlers Hotel in Colorado Springs.

This announcement has come to the Editor's desk from the Secretary of the American Board of Orthodontics, and should be noted carefully by all those expecting to appear before the Board.

School of Dentistry, University of Detroit

The School of Dentistry of the University of Detroit conducted an advanced course in orthodontics in accordance with the philosophy and technique of Dr. Charles H. Tweed, from July 7 to 21, 1946.

The course was under the direction of Dr. Samuel J. Lewis, and the faculty consisted of Drs. F. Copeland Shelden, Kansas City, Mo., Charles H. Tweed, Tucson, Ariz., Herbert I. Margolis, Boston, Mass., H. D. Kesling, La Porte, Ind., and Louis Braun, Detroit, Mich. Twenty-two of the class were returned servicemen, and 16 states were represented. The class consisted of the following men:

Robert M. Bailey, Boston, Mass.; David R. Berkson, Champaign, Ill.; Robert E. Coleman, Detroit, Mich; Gerard A. Devlin, Newark, N. J.; Leon Diskin, Detroit, Mich.; Arlo M. Dunn, Omaha, Neb.; George L. Englert, Chicago, Ill.; Marvin Goldstein, Atlanta, Ga.; Donald S. Graham, Seattle, Wash.; Joseph H. Grant, Detroit, Mich.; F. A. Grimmett, Youngstown, Ohio; Louis M. James, Jr., Kansas City, Mo.; Harry E. Jerrold, Brooklyn, N. Y.; Milton Lappin, Detroit, Mich.; Monroe E. Levin, New York, N. Y.; George R. McCulloch, Yakima, Wash.; H. H. Mueller, La Crosse, Wis.; A. S. Maxon, Walla Walla, Wash.; Willis H. Murphey, Fort Worth, Texas; Robert D. Payne, Phoenix, Ariz.; W. Glenn Phillips,

Jacksonville, Fla.; E. B. Pulliam, Corpus Christie, Texas; Denton J. Rees, Portland, Ore.; W. D. Reynolds, Chicago, Ill.; Louis W. Robinson, Youngstown, Ohio; C. A. Stratemann, New Braunfels, Texas; Louis F. Tinthoff, Peoria, Ill.

Army Dental Corps

The *Journal of the American Dental Association* has urged Congress to grant the Army Dental Corps administrative parity with the Army Medical Corps to provide "more efficient dental care" for Army personnel.

In the current issue, the *Journal* said that the A.D.A. has been forced to appeal to Congress because of the failure of the Army high command to correct administrative weaknesses in the Medical Department which seriously interfered with the work of dental officers during World War II.

A bill granting the Army Dental Corps administrative parity similar to that earlier granted by Congress to the Navy Dental Corps has been introduced in the House of Representatives by Congressman John J. Sparkman of Alabama.

The *Journal* said that enactment of the bill is necessary so that the work of the dental corps "will not continue to be hampered by the inept regulations, casual administration and glaring inequalities that provided such serious obstacles in the recent war."

The proposed bill provides that the Army Dental Corps shall be given authority to direct all matters relating to dentistry without the intervention of medical officers. The Dental Corps, however, is to remain as a part of the Army Medical Department under the command of the Surgeon General.

The *Journal* said that high army officers had repeatedly promised officials of the A.D.A. that administrative revisions would be made regarding the status of the dental corps.

The A.D.A. publication listed excerpts from correspondence between Dr. Carl O. Flagstad, of Minneapolis, chairman of the A.D.A. committee on legislation, and high army officers, including Surgeon General Norman T. Kirk, extending back to March, 1945.

This correspondence, the *Journal* said, reveals the Army record to be "a story of unfulfilled promises, unwillingness to support corrective legislation, hampering reaction, and open ineffectiveness."

"Returning dental officers almost without exception," the *Journal* continued, "confirm and enlarge upon the charges of inadequacy and inequality that are familiar to every dentist who served in the armed forces."

New York Society of Orthodontists

The next meeting of the New York Society of Orthodontists will be held at the Waldorf-Astoria Hotel, New York, on Monday and Tuesday, Nov. 4 and 5, 1946.

Pacic Coast Society of Orthodontists

The Pacific Coast Society of Orthodontists will hold its next general meeting in San Francisco, California, Feb. 20, 21, and 22, 1947.

J. CAMP DEAN, 1624 Franklin Building, Oakland, Calif., President.

Note of Interest

Dr. Ralph Olds Leonard announces the resumption of his duties at Culver Military Academy, Culver, Indiana, after service with the United States Army. Practice limited to orthodontics.

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The AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY is the official publication of the American Association of Orthodontists and the following component societies. The editorial board of the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY is composed of a representative of each one of the component societies of the American Association of Orthodontists.

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In the January issue each year, the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY will publish a list of all of the orthodontic societies in the world of which it has any record. In addition to this, it will publish the names and addresses of the officers of such societies.